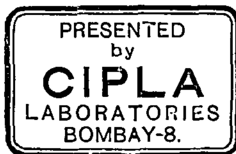
Roentgen Diagnosis *of the Heart and Great Vessels*

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First American Edition
new enlarged revision

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Foreword

This book requires no introduction to most roentgenologists. They received its previous editions favorably and quote them frequently. Many internists, cardiologists, thoracic surgeons, and physiologists as well as roentgenologists to whom the original volume was not available repeatedly expressed a desire to study the entire text if it could be made available in English. To satisfy these requests the author consented to this translation which, we hope, accurately presents the views of the author. Interpolations and annotations which mar many translations have been studiously avoided. The author selected an internist with a special interest in cardiology rather than a roentgenologist as a translator in the hope that the final text might more nearly meet the requirements of the various related medical and surgical specialties. Fortunately this additional safeguard proved unnecessary.

We have called this the first American edition Four years have elapsed since the appearance of the last German edition. During this interval considerable progress has been made in the roentgen diagnosis of diseases of the heart and great vessels. The contributions made by angiocardiography, aortography, electrokymography, and tomography partly reflect these advances Developments in cardiac surgery have posed new questions. Cardiac catheterization, measurement of pressures in the different chambers of the heart, and the analysis of blood gases indicate progress in intimately related fields and now form an indispensable part of cardiovascular roentgenology. Additional information has been obtained about pulmonary arteriovenous shunts and new conceptions have developed concerning the effects of systolic and diastolic overload on the single chambers of the heart. The inclusion of many of these topics in the present volume suggested the propriety of designating it as the first American edition although it is based essentially on the German edition of early 1949.

To incorporate these advances, revisions have been necessary, particularly in the sections on congenital malformations of the heart and great vessels. Many new illustrations had to be introduced. For absorbing this unanticipated additional expense, our thanks are due to Mr. Henry M Stratton of Grune & Stratton who made this possible. Many of the older illustrations are not tonal reproductions of films; that is, the heart appears in black instead of in white. The opposite practice is now standard. Apart from the impracticability or impossibility of locating the original materials, new reproductions would have increased costs to a point where the translation would not be generally available.

The shape of the heart, roentgenologically considered, is the morphologic expression of its function, and deviations from its normal function may result in alterations of its form. Consequently, roentgen findings must be integrated with the normal and pathologic function and with the clinical phenomena presented by the

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The Editorial Staff of Grune & Stratton rendered invaluable assistance. Although several roentgenologists read and criticized my translation, I am particularly grateful to Dr. Frank J. Borrelli, Director of the Department of Roentgenology, The New York Medical College, Flower and Fifth Avenue Hospitals, and Dr. Rito V. Grieco of the same department for detecting many minor errors of translation. Miss Natalie Pearlstein of the Art Department of the same institution made drawings of some congenital malformations from the original sketches of Professor Zdansky. For the index, which is more detailed than the original, I am indebted to Dr. Arthur J. Grossman, Department of Medicine, The New York Medical College, Flower and Fifth Avenue Hospitals. Miss Margaret C.Wheeler prepared the manuscript in its initial as well as final stages.

The translator was exceedingly fortunate in having the unstinting aid of Dr. David Scherf, Associate Professor of Medicine, The New York Medical College, Flower and Fifth Avenue Hospitals. Having been intimately and equally associated with Professor Zdansky and the translator for a period now reaching over thirty years, he was thoroughly familiar with the ideas of Professor Zdansky. If the translation more nearly approaches the true sense of the original in many places, this may be credited to its careful study by our mutual friend, Dr. David Scherf. For this the author and translator are deeply grateful. If, despite all these precautions, errors have occurred they are the responsibility of the translator.

Linn J. Boyd

New York
April, 1953

circulatory organs. To this extent, one may be justified in speaking of a functional roentgen diagnosis of the heart and circulation. One should, however, fully appreciate that the roentgenogram is able to reveal only coarse morphologic changes and that these alterations often are not pathognomonic for a single disease. Ambiguity of roentgen findings is inevitable because the possibilities of cardiac reaction are comparatively limited in contrast to the multiplicy of abnormal dynamic conditions within the heart and myocardium, pathologic situations in the systemic and pulmonary circulation, and disturbances in vasomotor regulation.

For these reasons, interpretation of roentgen findings is possible only in conjunction with the data provided by physical, laboratory, and electrocardiographic examination Correlated in this way, roentgen examination provides highly important insight into the functional and anatomic status of the heart.

An indispensible prerequisite for understanding roentgen findings is a thorough knowledge of the normal anatomic relations of the heart and other mediastinal organs. Accordingly, considerable space has been allotted to the roentgen anatomy of the mediastinum in this volume. The same situation prevails in respect to a host of other subjects, as exemplified by the influence of age, sex, body habitus, position of the body and the diaphragm, the effect of respiration, of the introduction and loss of fluids, of cardiac rate, and other factors.

Perhaps allusion to two other subjects may be permissible. Particular care has been paid to fluoroscopy of the heart and aorta which, when painstaking, richly rewards the examiner. Therein the author reflects, in the opinion of the translator, the dictum of Wenckebach: "sometimes a moving picture shows more than a post card." The second subject would appear to be particularly timely and informative· the influence of single strenuous as well as long continued efforts upon the heart from the roentgenologic standpoint. The observations should prove very informative for those who deal with athletes as well as those advancing expert opinions in connection with civilian or military claims

The section on hypertrophy and dilatation of the various chambers of the heart should prove very informative to many readers. It bridges the transition from the sections on the normal heart to those dealing with abnormalities

Naturally, most of the volume is devoted to the roentgen diagnosis of diseases of the heart and great vessels. Since these sections are, in the opinion of the translator, equally comprehensive and authoritative, reference to them may be omitted in this foreword.

It has been impossible in a book of this scope to cite the entire literature in detail. In the bibliography, an attempt has been made to select the most important sources and those containing further references. In this way, the book retains the character of a text book as well as that of a reference book. An endeavor has been made to bring the bibliography up to the end of 1952

The translator is deeply grateful for the assistance of many individuals, all of whom cannot be mentioned in this place. Among them are the following: Professor Zdansky permitted the translation to be made and offered many suggestions to improve it, I trust no serious errors escaped our combined scrutiny; the consent and cooperation of Springer Verlag (Wien), publishers of the original, should be mentioned. Thieme Verlag (Leipzig) kindly gave permission to include four color plates.

Contents

—Chapter One—

General Technic of Examination

I. Fluoroscopy, Orthodiagraphy, and Teleroentgenography

The essential part of the roentgenologic examination of the heart and great vessels is painstaking fluoroscopy. When conducted in many planes, fluoroscopy alone gives a plastic conception of the size and shape of the heart and its parts, reveals the relative position of abnormal structures to the heart and great vessels, and permits recognition of pulsations and movements which are vitally important in interpreting the morphologic findings.

It is advisable to begin examination with the patient standing or sitting There are several reasons why the erect position is ordinarily preferred. It is much easier to rotate an upright patient around his long axis. The difference in brightness between the mediastinum and lung fields is greater in upright than in recumbent patients because the lungs expand better, moreover, in the dorsal recumbent position, pleural effusions, so often present, spread over the posterior parts of the pleural cavities, rendering the lung fields somewhat dark. The erect position favors descent of the diaphragm, whereby the cardiac shadow is demarcated more sharply from the abdominal shadow Finally, many cardiac patients are able to stand or sit for a short time but cannot lie flat

On the other hand, *fluoroscopy in the horizontal position is* necessary *in* many patients and for the solution of special problems Some patients are unable to stand or sit, determination of cardiac size or of aortic diameter under optimal static conditions of filling should be undertaken in recumbent patients and with vertical passage of the ray. To test lateral mobility of the heart, fluoroscopy should be done with the patient in the right and left lateral positions Finally, a lateral view or the anteroposterior cardiac diameter of the recumbent patient with the ray projected from right to left may be of interest.

Fluoroscopy in different planes and positions selected according to the special features of each case and the particular problem, plus careful observation of the pulsations and the course of respiratory movements, provides, in most cases, the insight into the morphology and dynamics of the heart and circulation obtainable by roentgen examination Only in a few patients and for special diagnostic reasons are films required, or do kymographic or cinematographic records seem desirable.

The correct picture of the greatest expanse of the chest organs is obtained only by parallel projection (fig. 2) and this is most nearly approximated by orthodiagraphy or teleroentgenography.

Cardiac orthodiagraphy is the graphic registration of the parallel projection of the heart. The resultant orthodiagram represents the greatest expanse of the heart in the direction parallel to the plane of projection.

Originally, Moritz constructed an orthodiagraph for the heart with the tube and screen firmly connected (fig. 3). At the site of the central ray there was a small aperture which appeared as a black dot on the illuminated screen. If the screen and tube were moved so that the dot indicating the central ray travelled along the

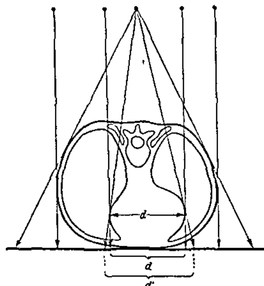


FIG 2—Comparison of central and of parallel projection, d = true largest diameter of the heart, d' = diameter of the cardiac shadow with central projection.

silhouette of the cardiovascular shadow, the contour could be marked, point by point, directly on the skin of the subject through the aperture or, even better, on a sheet of paper behind the screen

G Schwarz greatly simplified the technic. With his method, no special contrivance is necessary as long as the movements of screen and tube are independent of each other. Orthodiagraphy can be performed with any apparatus satisfying this condition. The basic principle is as follows: a small central beam is cut out of the divergent rays emerging from the tube by narrowing the diaphragm, and the borders of the cardiovascular shadow are registered with this beam (fig. 4)

With the shutter properly closed, a small light field, 3 to 4 cm in width, appears on the screen, produced by rays around the central ray. The center of this field on the screen corresponds to the central ray. With this beam, composed of almost parallel rays, the silhouette of the cardiovascular shadow is recorded. The screen, held vertical in front of the chest, is locked and the tube is shifted so that the center of the light field passes along the edges of the cardiovascular shadow. To provide

For permanent records, for precise measurements and for exact comparisons, neither fluoroscopy nor copies of the outlines of the cardiovascular borders under ordinary conditions of fluoroscopy suffice, this also holds for films obtained with the technic for routine radiography of the lungs With the customary target distances of fluoroscopy and ordinary teleroentgenography of the lungs, rays penetrate the body divergently, according to the laws of central ray projection, the image of the cardiovascular complex and thoracic walls projected on the screen or film is magnified. The picture is by no means a simple linear enlargement of parallel projection since different intrathoracic structures, according to their lateral expanse and their

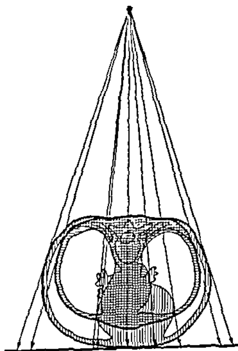


Fig 1 —Central projection of two cross-sections of the same thorax. Owing to differing sizes of single parts and their varying distances from the plane of projection, *the extent of magnification* differs.

distances from the tube on one hand, and the plane of projection on the other, experience varying magnification. Moreover, rays penetrating the body divergently do not pass tangential to the points most lateral on the cardiac surface but to points near the posterior cardiac wall, therefore the latter exert a determining influence on the form in the roentgen picture (fig. 1).

With a structure as irregular in contour as the cardiovascular complex is, mathematical constructions of the parallel projection *from the picture of the* central ray are not accurate; accuracy can be achieved by the equation $O = Ba/b$ only with bidimensional or regular objects. In this formula, O is the true size of the object, B, the size of the object image, a, the target-object distance; b, the target-screen distance. At best, *this formula* is applicable to determining the true diameter of the aorta.

The maintenance of identical conditions, as far as possible, in the preparation of orthodiagrams is unconditionally indispensible if they are to be compared.

The following points should be observed:

1. Orthodiagrams must always be recorded with the body in the same position; the given patient must always be erect or recumbent, for cardiac size and shape depend to a great extent on the position of the body (p 90). Since roentgen examination of the heart, as a rule, is conducted with the patient upright, it is well to secure orthodiagrams also in this position (vertical orthodiagram). For special reasons and in some patients it may be desirable or even necessary to outline the cardiac silhouette in the recumbent position (horizontal orthodiagram). To decide, however, whether and to what extent the heart has changed its size and shape during the period of observation, it is absolutely necessary to retain the erect or horizontal position for this particular patient.

2. All orthodiagrams must be recorded in the same phase and, as far as possible, with the same depth of respiration. Respiration exerts considerable influence on the size and shape of the cardiovascular complex. As a rule, the expiratory phase of ordinary quiet breathing is preferred. The extreme phases of respiration are intentionally avoided since breaths differ greatly in depth in patients with normal hearts and even more when circulatory disease is present.

Since, with quiet breathing, expiration normally causes lateral spread of the mediastinal shadow, the orthodiagram recorded at this time represents the widest mediastinal shadow obtainable during quiet respiration.

3 The pulsations along the borders of the mediastinal shadow require consideration. The rhythmic in- and outward movements make it necessary to record the individual points always in the same phase of motion Those points along the borders of the mediastinal shadow are noted where the pulsations reach the most lateral point (Moritz). Consequently, the arc of the left ventricle is recorded at the end of diastole and the left border of the vascular shadow at the height of systole. On the other hand, the right cardiac border is determined in diastole or in systole, depending on whether and to what extent it shows ventricular or atrial pulsations. Since, under these circumstances, all marginal points of the shadow are not recorded in an identical phase of the cardiac cycle, the resultant figure has a size and shape which does not correspond to reality at any given moment; this constitutes the chief difference from an instantaneous teleroentgenogram.

While this procedure may seem somewhat complicated at first, actually it greatly simplifies registration. Thus, in orthodiagraphy, it is not necessary to determine whether a marginal point just recorded attained its lateral point of reversal under the influence of respiration, of systole, or of diastole; rather, one registers each point at the moment the maximum lateral distance is reached.

4 Although the orthodiagram is, to a certain extent, mechanical registration, it demands thought and judgment. Gross mistakes are avoided only when the record is made after careful preliminary fluoroscopy. Moreover, at frequent intervals during registration the shutter must be partly opened momentarily to remain oriented. This is particularly necessary when the lung fields and hilar shadows are abnormally dark, when large hearts extend far to the left, and especially to outline the cardiac apex which may be difficult to see even when the heart is *normal* (p

the orthodiagraphic silhouette of the cardiovascular complex, the marginal points are marked with a glass pencil on the screen as they appear in the center of the light field

Some contend that this procedure is less exact than ordinary orthodiagraphy. We do not share this opinion because the physical presumptions of the Schwarz method are correct. The subjective factor in precise focussing of the cardiac silhouette in the central ray is also unavoidable in the orthodiagraphy of Moritz. The notion that the method is conducive to hastiness and carelessness is hardly a valid

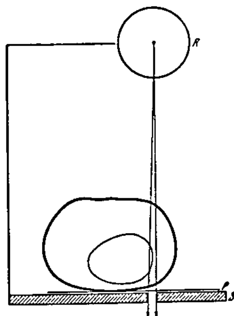


Fig 3

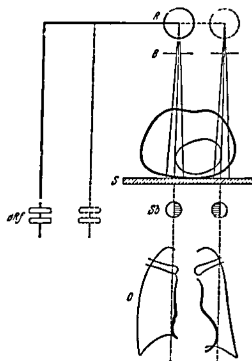


Fig. 4

Fig. 3 —Diagram of the Moritz orthodiagraph R = tube, S = screen, P = paper.

Fig 4 —Schematic representation of orthodiagraphy by the Schwarz method R = tube, B = tube shutter, BRf = diaphragm and tube control, S = screen, Sb = screen image, O = orthodiagram

objection against the procedure itself for strict observance of rules and exact work are prerequisites of every medical act; inaccuracies arising from disregard of these essentials should not be charged against the method Naturally, training is necessary to secure satisfactory results but this holds equally for all orthodiagraphic records.

The maximal margin of error which we calculated with orthodiagraphy after the Schwarz method does not exceed that of the original orthodiagraph (Dietlen, Hammer). This amounts approximately to ± 3 mm. for the transverse diameter of the heart when one method is used immediately after the other without the subject performing any physical exertion in the interim This condition must be fulfilled because the actual size of the heart varies.

The rules of orthodiagraphy must be followed closely in teleroentgenography. The films must always be taken with the body in the same position, in the same phase of respiration, and, when possible, at the same time of day and when fasting. The position of the tube must always be the same in respect to the body and to the film; it is best to focus on the spinous process of the sixth thoracic vertebra and on the center of the film To avoid small distortions, lead markers on the front and back of the chest are brought into line. Although it might seem advisable to take the film in deep inspiration to secure better contrast, this should be avoided owing to the danger of involuntarily inducing a Valsalva experiment, it is better to suspend respiration for the period of exposure when the patient has been breathing quietly (Dietlen, Groedel, Hammer, Assmann).

Even when all these precautions are taken, teleroentgenography possesses a real source of error arising from pulsations of the cardiac shadow. To eliminate them, devices have been contrived to send the current through the tube by mechanical or electrical transmission of the pulse wave (*Eijkman, Weber, v. Elischer, Huismans*, Cottenot, Strauss, Ludwig) or by the action current of the heart (Bergk and Chantraine, Hirsch and Schwarzschild). Greater exactitude is obtained since the film is always synchronized with the same phase of the cardiac cycle.

The advantages and disadvantages of orthodiagraphy versus teleroentgenography have often been weighed against each other. Teleroentgenography minimizes subjective error and spares the investigator from unduly prolonged exposure to roentgen rays. Entirely apart from the greater expense of teleroentgenography, it is indisputable that careful orthodiagraphy by an experienced observer is more exact for the determination of the absolute size of the heart Even when the target-film distance is as great as 2 to 2.5 meters the rays still pass through the body with appreciable divergence, consequently they enlarge and distort the image of the cardiovascular complex. The transverse diameter in a cardiac teleroentgenogram of an obese individual in whom the heart-film distance is relatively great or when the heart is enlarged, may appear several centimeters greater than in an orthodiagram (Hammer) Furthermore, demarcation of the cardiac apex is much more difficult with teleroentgenograms than with fluoroscopy or orthodiagraphy Consequently Moritz and Dietlen were probably correct in constantly advocating orthodiagraphy. Moreover Groedel, Hammer, Assmann, Eyster, and Arkussky expressly state that orthodiagraphy is preferable to teleroentgenography when accurate comparative measurements are required for investigations of the influence of various agents on cardiac size

II. Plastic Reconstruction of the Heart by Means of X-rays

Early attempts were made to reconstruct the heart in its natural size and shape by means of x-rays. The plastic model of this kind, produced by Moritz, naturally was very imperfect for it was constructed simply from a sagittal and frontal orthodiagram Palmieri was the first to develop a relatively simple method making it possible to reconstruct the heart from a series of selected projections.

22) To outline the cardiac apex it is advantageous to have the patient breathe deeply several times while the shutter is partly open, usually this creates greater contrast between the border of the heart and the lung fields, lightened by inspiration, on the one hand, and by the dark abdominal shadow, on the other. As stated earlier, the orthodiagram is not recorded during deep inspiration nor with the shutter open, but in the expiratory phase of quiet respiration with the shutter drawn closely.

5. Special attention should be devoted to painstaking avoidance of slight, unintentional rotation of the patient around his long axis as well as to a varying forward or backward inclination of the upper part of the body, even slight changes of posture profoundly influence the projected image. The ease with which this can be eliminated by having the patient recumbent constitutes the special advantage of this position (Moritz) Undesirable postural alterations of erect patients can, however, be reduced to a minimum, by insisting that both scapulae and both heels constantly touch the wall of the fluoroscope, as a further safeguard, lead markers may be attached to the anterior and posterior chest walls to ensure exact reproduction of the position (Schwarz, Weiss). We attach to lead crosses by adhesive, one over the spine of the fifth or sixth thoracic vertebra and the other over the sternum. When the tube focus centers on the posterior marker, the transverse and vertical limbs of both markers coincide exactly. This makes it easy to recognize and to correct lateral rotation and different inclinations of the body. Special contrivances for holding erect patients, as devised by Moritz, can be eliminated by following these instructions

6 To compare cardiac size exactly, the orthodiagram should be recorded as far as possible at the same time of day, perhaps in the morning before breakfast, and after rest. Differences in the filling of the stomach and bowel may exert considerable influence on the position of the diaphragm and exertion or excitement can alter cardiac size.

With dextrosinistral passage of the ray, great attention must be paid to the proper position, the least rotation around the long axis of the body causes great errors In the correct position with exact frontal passage of the ray, the sternal shadow is smallest, most intense, and sharply demarcated on both sides Lead markers on the anterior and posterior chest greatly facilitate correct posture in this position, they ensure the same inclination of the upper part of the body as with dorsoventral passage of the ray, an important factor in calculations of cardiac volume (p. 75)

Every orthodiagram should include a sketch of the contours of both clavicles, the domes of the diaphragm, the costophrenic angles and, sometimes, the spinal column as well. Like the mediastinal shadow, they are all recorded in expiration These outlines serve mainly as points of orientation for the recognition of displacements within the thorax. If the clavicles on two orthodiagrams are superimposed, the presence and extent of alterations of diaphragmatic level is immediately apparent, changes of this level strongly influence cardiac position and shape

Teleroentgenography of the heart was introduced by Kohler and Albers-Schonberg in an attempt to reduce the error of central projection as far as possible by increasing the target-film distance to 2 meters or more.

III. Roentgenkymography of the Heart and Great Vessels

Roentgenkymography describes the cardiac movements demonstrable by x-ray. In principle, the outlines of the cardiac shadow are projected through one or more slits cut into a holder impervious to x-rays By moving the film in a direction perpendicular to the slits, the marginal movements of the cardiac shadow are recorded on the film as a curve.

By virtue of this principle the possibilities as well as the limitations of the method become apparent.

Undoubtedly graphic registration of movements is vastly superior to the simple observation in fluoroscopy, one can detect small or volatile excursions invisible to our eyes. On the other hand, the movements executed by the borders of the cardiac shadow do not necessarily correspond in space to movements of superficial points of the heart. Actually tridimensional movements are shown in bidimensional projection. Consequently movements at other than a right angle to the path of projection are reduced and in extreme cases, especially when they run parallel to this path, may be completely erased.

During its action the heart undergoes many alterations of shape and position. Consequently pulsations at the edges of the cardiac shadow do not correspond to real volumetric changes of the cardiac segments forming the borders at that moment In drawing attention to the interference (Heckmann) between changes of volume and of position, recorded as movements at the edge of the cardiac shadow, Ludwig and Heckmann showed that these movements are uncontrollably magnified, reduced, abolished, or even reversed through interference so that double notches and other deformations of the curve can appear.

As a result of these pulsatory transformations and positional changes of the heart, the same marginal point of the cardiac shadow, the movement of which is observed or recorded, may be evoked sequentially by different points on the cardiac surface.

These brief remarks should suffice to indicate that neither kymographic records nor excursions perceived fluoroscopically, necessarily correspond to actual movements of points on the cardiac surface

Kymographic records have another source of error intrinsic in the procedure. The recorded curves correspond to actual excursions of a given marginal point only when the edge of the cardiac shadow is perpendicular to the slit of the kymograph If the edge of the shadow moves obliquely to the slit, the curve no longer exactly reproduces the excursions of a definite marginal point; other points move into the opening of the slit and participate in the registration (Zdansky and Ellinger, Cignolini)

These facts saddle roentgenkymography with uncertainty, necessitating great restraint in interpretation In a careful analysis, Ludwig denied that roentgenkymography was an exact method of functional investigation. Actually, its curves must always meet the test of other more precise observations on man and animals, contradictions arising must be assigned to imperfections intrinsic to roentgenologic observations of motion in general and to roentgenkymography in particular. At most

Palmieri's procedure had two steps.

1. *Films of the heart in selected diameters* were obtained, as many as were feasible, with a constant focal distance; the angle about which the subject was rotated around his long axis on the revolving chair was noted exactly for each film.

2. A plastic model of the heart was then prepared. at first, silhouettes of the cardiac shadow were cut from cardboard from each film taken at a different angle; in place of the heart a block of clay was rotated around its long axis, one end of a wire was fixed at the target of the x-ray tube, in place of the film one of the above mentioned silhouettes was placed, likewise at the same distance. If the wire was moved along the edge of the paper cut-out it cut a form from the clay block corresponding to the one existing at the moment As the block rotated each time at an angle corresponding to a given phase a plastic model of the heart gradually emerged. Since the wire was fixed at the site of the target and ran along the successive silhouettes, the model carved was formed by rays running tangential to the heart and reproduced its true size and shape.

Brednow altered this method by constructing his model from several tele-roentgenograms, the silhouettes of the individual pictures were cut from plaster of paris by a saw. The axis of the saw corresponded to the central ray A small light, fixed on the side opposite the film permitted one to follow the contours of the cardiovascular shadow. The resultant model was somewhat larger than the heart because the error of central projection with 2 meter teleroentgenography was not completely eliminated

Lysholm as well as Berg and Schatzki reported a one-step method Its principal difference consisted in joining the x-ray tube and cutting wire in such a way that the wire cut the cardiac form directly from the clay or plasticene during fluoroscopy as one moved the central ray along the silhouette. Since the tables on which the patient and the clay block were rotated turned synchronously, the wire gradually carved a plastic model from the block Fundamentally, the methods differ only in that with one the silhouettes are provided by telefluoroscopy (Lysholm) and in the other by orthodiagraphy (Berg and Schatzki).

These plastic models have only didactic value The beautiful models prepared by Belski for the American Heart Association prove this. For practical clinical purposes the technic has little application The procedures are so time-consuming that their employment for diagnostic purposes is extremely limited, this is particularly true when the model is cut directly during fluoroscopy The methods of Palmieri and of Brednow have the advantage of requiring only a short time to secure the films for preparing the model, consequently errors from possible alteration of cardiac size during preparation of the model are greatly reduced

Plastic models more nearly approximate the ideal of volumetric determinations of the living heart than calculations of cardiac size from one projection or another. Accurate demarcation of the heart from the great vessels and from the liver naturally is not possible in a cardiac model. Nevertheless Palmieri emphasizes that the plastic method provides a way for determining accurately the position of the cardiac apex

Plastic models made no important contribution to the precise analysis of the vascular complex.

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one may trust roentgenkymography when it confirms well-founded expectations and previous experience.

We cannot concur with the statement often expressed that kymography regularly assists in the diagnosis of some conduction disturbances and cardiac arrhythmias. A false premise is involved when roentgenologic methods are advocated to obtain facts available from thoroughly verified methods by whose very nature more

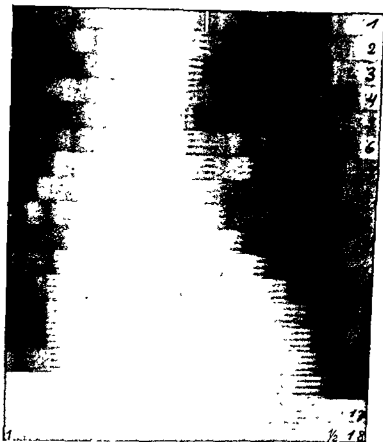


FIG 5.—"Step kymogram." Registration of pulsations on a moving film with a stationary grid (From Pl Stumpf, *Röntgenkymographische Bewegungslehre*)

exact results are secured. Such efforts are economically unsound and serve neither science nor the patient. Rather, they tend to bring roentgenology into disrepute. The diagnostic achievements of roentgenology in its own field are so enormous and uncontested that there is no reason for dabbling outside its own frontiers when other adequate, established methods exist.

These statements are not intended to imply that kymography lacks value and significance in some problems, provided that the results are critically analyzed, unfortunately this attitude has not always prevailed in the literature which has grown to vast proportions

Roentgenkymography was originally announced by Sabat in 1911. Independently, it was first employed for registration of cardiac movements by Gott and

Rosenthal. Subsequently Becker, Crane, Knox, Chamberlain and Dock, Stenström and Westermark as well as Laurell and Sundberg utilized it for the same purpose. Then, Hitzenberger and Reich further developed it for registration of diaphragmatic movements. Its employment became widespread, however, after Stumpf as well as Scherf and Zdansky used it for systematic investigation of the heart.

Considerable literature resulted from the discovery of surface kymography by Stumpf. A multiple slit grid is interposed between the heart and film so that many adjacent sections of the cardiovascular shadow can be simultaneously kymographed.

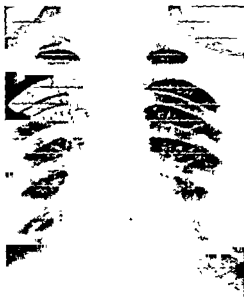




Fig 6 Fig. 7

Fig 6—"Surface kymogram" Registration of the pulsations on a stationary film with a moving grid

Fig 7—Kymogram of the right atrium and the left ventricle (long strip recording by Zdansky and Ellinger)

The film may advance over the stationary grid (step kymogram, fig. 5), or the moving grid may pass over the immobile film (surface kymogram, fig. 6). By these simple methods the reciprocal time relations of movements are determined from numerous segments of the cardiovascular shadow, previously, only single sections could be registered The movements of the grid of the surface kymogram at an optional speed create an illusion of motion (Stumpf) and undoubtedly this has merit in the illustrative method of teaching Opposed to these advantages, the curves with both types of kymograms are short, compressed, and reveal only a few details. Consequently, in their investigations Zdansky and Ellinger retained, with slight modifications, the classical long strip records (fig. 7); on the other hand Cignolini

as well as Delherm and his coworkers constructed devices which made it possible to focus selected portions of both cardiac contours in a system of interchangeable slits so that movements in a section of interest could be recorded on long films (fig. 8).

Electrokymography represents another method for registering the pulsations of the cardiovascular shadow. Originally, in 1936, Heckmann employed a photoelectric cell whose current was supplied to a recording device through an amplifying tube; it registered in a curvilinear manner the differences in translucency which developed on the screen from pulsations of the heart. Subsequently, photoelectric registration



Fig 8.—Kymogram of the cardiovascular shadow (long strip recording with multiple slit method of Cignolini).

of the pulsations found wider acceptance, at first through the work of Lian and Minot but particularly as the result of improved technic by Henny and Boone as well as Luisada and Fleischner. For the analysis of the resultant curves, simultaneous registration of the phonocardiogram proves most suitable

Although fundamentally mechanical and electrical kymographic registration of movements of the cardiac borders must lead to the same results, electrokymography

and it is essentially cheaper in operation and cost than mechanical roentgenkymography. "Phase kymography" recently introduced by Heckmann seems to open new diagnostic possibilities.

The results obtained by roentgenkymography are discussed in the individual

IV. Angiocardiography

By means of angiocardiography the cardiac cavities and the great intrathoracic vessels can be roentgenologically demonstrated. In combination with the electrocardiogram (Sussman and Grishman, Lind and Wegelius) and particularly in the form of cineangiocardiography (Janker) it affords an incomparable insight into the inner dynamics of the heart (p. 62). It reveals the functional alteration of the size, shape and reciprocal spatial relations of single cardiac chambers during cardiac activity (Zdansky, Lind and Wegelius).

After Forssmann (1929), Moniz, Carvalho, and Lima (1931) as well as Castellanos, Pereira, and Garcia (1927) attempted contrast filling of the heart and pulmonary vessels in living people more or less successfully, angiocardiography was developed into a routine method of great diagnostic value by Robb and Steinberg (1936–37). This became possible when opacifying agents became available which were relatively nontoxic despite high concentrations and the injection of large amounts. At present, Diodrast or Uroselectan B (Neo Iopax, Perabrodil, Joduron, or Umbradil) is employed in a 70 or 75 per cent solution, respectively. Nevertheless, all these high molecular and hypertonic iodine preparations are not completely innocuous. Intravenous injection may result in a fall of blood pressure with an increased pulse pressure amplitude, an increase of venous pressure, change of cardiac rate, arrhythmia, local vascular spasm, reduction of tonus in vessels supplying large areas, a sense of oppression, cough, headache and nausea as well as a transient urticaria, loss of consciousness, respiratory standstill, and pulmonary edema In recent years, fatalities after angiocardiography in the United States, Canada, Great Britain, and Sweden have averaged 0.38 per cent (Dotter and Steinberg) Death may occur in minutes, hours, or even after a few days. These disastrous results have occurred chiefly in patients with cardiac decompensation and severe cyanosis or on reinjection of the contrast material at short intervals in the progress of an examination

Consequently, individuals with large hearts and congestive heart failure must be excluded from angiocardiographic examination, the injection should not be repeated on the same day, prior to the injection 2 drops of the agent should be instilled into the conjunctival sac and 1 to 2 cc. injected intravenously in order to ascertain whether there is hypersensitivity to the agent, children and very excited adults should receive sedatives in advance As far as possible individuals with allergy should not be subjected to examination.

For preparation of the patient, barbiturates orally or rectally administered, as well as a mixture of methadon, ephedrine, and scopolamine subcutaneously are available General anesthesia is necessary only with very restless patients.

The amount of contrast agent injected must be sufficiently large to secure a good demonstration of the cardiac cavities but not so great that the danger of injury exists. We give approximately 1 cc. per Kg. body weight In children of 2 to 3 years we use 15 to 25 cc., from 4 to 14 years 25 to 40 cc in children over 14 up to 50 cc , and in adults 50 to 70 cc. For aortography 10 to 20 cc. suffices.

If these precautions are taken, practically no serious incidents are encountered. Wegelius had no serious complications in about 250 cases and Zdansky *none* in almost 100 Nevertheless, angiocardiography should be undertaken only when all

other diagnostic measures have been exhausted and when one must decide, whether to operate and what surgical operation should be undertaken.

To protect the investigator from undesirable radiation, Morgan devised an injector whose piston is pressed by air pressure, a protective screen, through which the arm of the patient is thrust, also suffices.

To demonstrate the cardiac cavities, the cubital vein is exposed and the contrast agent is injected through an attached, wide cannula within 1 to 2 seconds. The first film should be taken after the injection of the first 5 cc. For aortography a catheter is advanced through the exposed right brachial artery to the middle of the ascending aorta, the injection is made by means of a pressure syringe

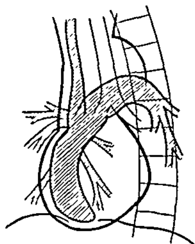


Fig 9

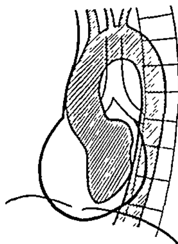


Fig 10

Fig 9 —Angiocardiogram of the right heart and pulmonary artery with its branches in the left anterior oblique position

Fig 10 —Angiocardiogram of the left heart and aorta with its brachiocephalic branches in the left anterior oblique position

We undertake the examination basically in recumbent patients with passage of the ray optimal for the individual case.

To demonstrate the ventricles, sagittal passage of the ray and the left anterior oblique position is recommended. In the latter, the position of the ventricular septum, the trunk of the pulmonary artery, the left pulmonary artery, and the course of the thoracic aorta are well represented Figures 34 and 35 illustrate the contrast-filled right (dextrogram) and left (levogram) heart with sagittal projection and figures 9 and 10, the left anterior oblique position

It has proven very advantageous to secure synchronous roentgenograms in two planes at right angles to each other (Chamberlain and coworkers, Axén and Lind). Fredzell and coworkers combine this technic with direct serial roentgenography which permits an exact analysis of the normal and pathologic heart. It is particularly worthwhile to obtain the proper electrocardiogram with the film (Sussmann and

Grishman, Morgan, Lind and Wegelius, Dotter and Steinberg) because unawareness of the phase of the cardiac cycle in which the film was obtained may lead to serious mistakes. Thus, systolic narrowing of the outflow tract of the right ventricle occasionally has simulated a stenosis of the pulmonary infundibulum (F. J. Hodges). With cineangiocardiography (Janker), this danger does not exist, for then the dynamics of the heart are reproduced continually in the film.

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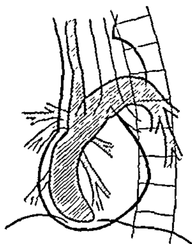


Fig. 9

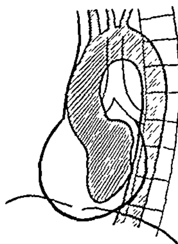


Fig 10

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from the left or right in front to the right or left behind, respectively, the views are called the second posterior and the first posterior obliques.

Holzknecht, Kraus, v. Criegern, Moritz, Rieder, Rosenthal, Th. and F. Groedel, Schwarz, de la Camp, Weinberger, Dietlen, and Assmann in particular made outstanding contributions to the interpretation of the normal roentgenogram of the heart in different positions.

1. The Anterior View of the Heart in the Erect Position (Figure 12a and b)

Fluoroscopic examination begins in the anterior position with dorsoventral passage of the ray. In this position the mediastinal shadow (Holzknecht) is almost homogeneous, this shadow is located between the lung fields and is formed by the mediastinal organs and structures, the spine and the sternum. Of these, the trachea descends as a translucent band in the midline of the neck or somewhat to the right and it can be followed to the lower borders of the clavicles. The deeper parts of the trachea, the bifurcation and the two main bronchi can be recognized, under the usual conditions of fluoroscopy and in teleroentgenograms, only in thin subjects and in children. The right main bronchus in particular often is visible as a clear band descending obliquely from the midline above to the right cardiovascular angle. Adjoining its right border, frequently one sees a spindle-shaped shadow, the size of a pumpkin seed (fig. 13) which corresponds to the azygos vein (Busi, Ottonello), it emerges from behind at the right tracheobronchial angle to pass toward the superior vena cava. When venous pressure increases, owing to right heart failure or from inflow stasis in constrictive pericarditis, the azygos shadow occasionally enlarges considerably (Durieu and Lequime). Cephalad, it continues in a linear shadow adjacent to the right wall of the trachea and produced by the mediastinal pleura seen on edge.

In children, Fanconi and Wechsler described two medially convex, thin stripes running craniocaudad within the clear tracheal band and often nearly touching each other in the midline Danelius recognized them as the shadows of the right and left vertebromediastinal pleura which are almost in the midline behind the esophagus, at this place the pleural cavities are almost in contact (fig 13)

In this position the vertebral column is almost invariably visible as a dark median shadow In addition to serving as an important landmark it should be observed carefully since a slight curvature, easily overlooked during a casual physical or roentgenologic examination may explain some alterations of shape (Roesler) of the cardiac shadow (p. 252) or an otherwise inexplicable or even misleading inspiratory mediastinal wandering (Zdansky) (p. 252).

As a rule, the right border of the mediastinal shadow is composed of two convex arcs They are about equal in length. The lower arc is rounder, more opaque, and projects beyond the right vertebral border about twice as far as the upper, flatter, more translucent arc which may ascend almost in a straight line. Real deviations in the length and extension of the arcs to the right often depend upon pathologic alterations of the heart or the vessels, providing there is no thoracic deformity and no shrinking or space-occupying process in the lungs or pleura.

The lower arc, the right cardiac border, is formed largely by the right atrium,

Chapter Two

The Normal Heart

I. The Roentgenogram of the Normal Heart

To obtain a plastic image of the shape of the heart and the size of its single sections, fluoroscopy in differing positions is necessary (Holzknecht, Weinberger). In 1909 Hoffmann suggested designating the different positions by the number of degrees through which a patient was rotated to the right about his long axis, starting with dorsoventral passage of the ray to reach the particular position (fig 11) In position 90 for example, the rays penetrate transthoracic from right

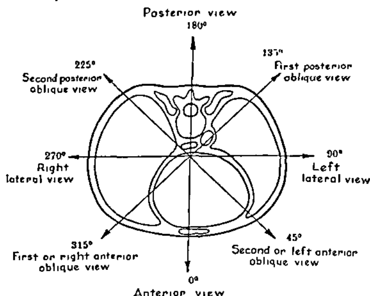


Fig. 11.—Passage of ray in different typical positions of fluoroscopy

to left. At present, the positions are usually called anterior or posterior, right or left lateral depending upon whether the breast, back, the right or left side of the patient is turned toward the screen. In addition, fluoroscopy in oblique positions (Holzknecht) has special importance. In the first or right anterior oblique (fencing) position the rays pass through the body from left behind to the right in front. The second or left anterior oblique (boxing) position is secured by rotation to the right so that the rays pass from the right behind to the left in front. When rays penetrate

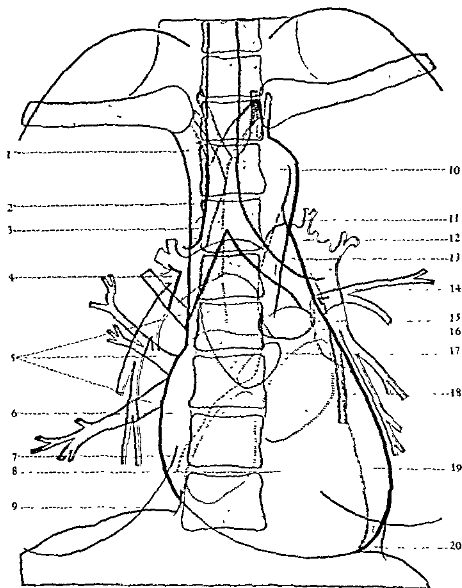


FIG. 12b — Anatomic analysis of the anterior view

1 V brachiocephalica (innominate vein) dext.
2. V cava *superior*
3 V thoracica longitudinalis dext (V azygos)
4. Right pulmonary artery
5 Right pulmonary veins
6 Right atrium
7. Projection of tricuspid ostium
8 Right ventricle
9. V. hepatica dext
10 Aortic arch
11 Lig arteriosum
12 Left pulmonary artery
13 Trunk of pulmonary artery
14 Left pulmonary veins
15 Projection of pulmonary ostium
16 Left *atrium*
17 Projection of aortic ostium
18 Projection of mitral ostium
19 Left ventricle
20 Fat pad



Fig 12a —Anterior view.

in the supradiaphragmatic section, however, the right ventricle may form some of the edge

Opinions on whether, and to what extent, the right ventricle contributes to the formation of the right cardiac border were and still are not in full agreement. According to F. M. Groedel, de la Camp, Zehbe, Assmann, Dietlen, and Vaquez and Bordet as well as Laubry and his coworkers, the right ventricle does not reach the right cardiac border. Assmann, Dietlen, and Vaquez and Bordet allow an exception: if the heart is perpendicular, owing to a low diaphragm, the right ventricle can

seen on the diaphragmatic boundary of the dextrogram, medial to the right costodiaphragmatic angle.

The right cardiac border joins with the right dome of the diaphragm at a sharp angle, the right cardiodiaphragmatic angle. Frequently this angle is bridged by a pale shadow which extends laterad and downward; this shadow, which is not invariably present, may first appear or may become more apparent with inspiratory descent of the diaphragm The shadow is created by the right hepatic vein, for the upper edge of its orifice in the inferior vena cava usually projects above the diaphragm (Hasse, Elias and Feller, Hitzenberger). It should not be confused with adhesions which are common in the cardiodiaphragmatic angle; the vein can be distinguished easily since normally the median section of the diaphragm bends caudad without hindrance during inspiration whereas it is delayed and becomes stretched when it is fixed by adhesions in the cardiodiaphragmatic angle. Rarely, a similar shadow may be created by a collection of fat between the parietal leaf of the pericardium and the mediastinal pleura (Assmann, Herrnheiser, Kautz and Pinner).

Sometimes—particularly in elderly emaciated individuals with a low diaphragm and a median heart and not rarely even in children—a shadow runs obliquely upward and outward within the right border of the heart, it continues in a vascular shadow running horizontally or downward within the base of the right lung field It corresponds to the right lower venous infundibulum of the left atrium into which the corresponding pulmonary veins empty

The edge of the slightly convex or perpendicular shadow rising above the right cardiac border, represents the right boundary of the vascular band. As a rule, it is formed by the right edge of the superior vena cava or, more rarely, by the ascending aorta (Holzknecht, v. Teubern, Frik, Delherm and Chaperon). Cephalad, this arc joins a lighter shadow bending concavely which can be followed up to the clavicles, it represents the right border of the V. brachiocephalica (anonyma) dextra. Since the right vascular border is usually convex and frequently shows, on the screen, a distinct outward systolic pulsation, many have suggested that at this place the ascending aorta rather than the superior vena cava forms the edge Catheterization of the superior vena cava in living subjects by Forssmann and angiocardiography, however, clearly reveal that the superior vena cava tends to project farther to the right than the ascending aorta in both living subjects as well as in cadavers Moreover, not rarely, the ascending aorta is noted as a darker band within the right vascular border and is seen to bend median at the level of the bifurcation. The convex course of the caval shadow to the right, commonly observed, depends upon the close application of the thin-walled vein under low tension to the taut thick-walled ascending aorta which is under high pressure, as the aorta curves convexly to the right, the vein follows its course An outward systolic pulsation of the right border of the vascular shadow, if seen, does not prove that the aorta projects beyond the superior vena cava (p. 60)

Although in most cases the superior vena cava undoubtedly forms the right border of the vascular shadow, as patients grow older the aorta, owing to widening and elongation, is more likely to project beyond the vein (Schwarz, Vaquez and Bordet, Rosler, Holzmann, Laubry and others).

The left border of the mediastinal shadow has two strong convex arcs between which there is an indentation called the cardiac waist.

appear on the right cardiac border in the supradiaphragmatic area (fig. 54). Under these circumstances, the heart rotates around a vertical and sagittal axis so that its ventral and diaphragmatic borders are situated more to the right (Schwarz). On the contrary, Holzmann maintained that even normally the right ventricle occasionally contributes to the right border for a short distance. In this opinion he has the support of some anatomists (Tiedemann, Merkel, Rauber-Kopsch, Schultze-Lubarsch,

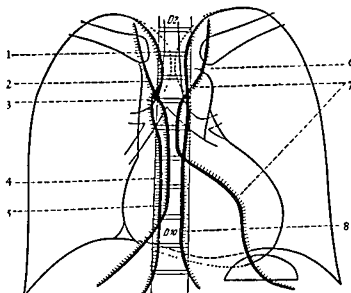


FIG 13 —Course of the costo- and the vertebromediastinal folds of reflection of the pleural cavities At the level of the upper thoracic segments, the pleural cavities approach each other behind the esophagus so closely that they are separated only by the reduplication of the pleura (dotted lines). The V thoracica longitudinalis dextra (azygos vein) is embedded in the right tracheobronchial angle. In the roentgenogram it often appears as a shadow with the shape of a pumpkin seed.

1. Fold of reversal of the right vertebromediastinal pleura
2. Fold of reversal of the right costomediastinal pleura
3. Azygos vein
4. Fold of reversal of the right vertebromediastinal pleura
5. Fold of reversal of the right costomediastinal pleura
6. Fold of reversal of the left vertebromediastinal pleura
7. Fold of reversal of the left costomediastinal pleura
8. Fold of reversal of the left vertebromediastinal pleura

Tandler). Laubry and coworkers, however, never saw the right ventricle extend as far as the right cardiac border when contrast agents were injected into the cadaver heart without opening the thorax, this does not preclude the possibility that the outer wall of the right ventricle can contribute to the formation of the right cardiac border

In the angiocardiogram as well, the right ventricle does not attain the right cardiac border with normal position of the diaphragm and particularly in the recumbent position Thus, the indentation of the atrioventricular junction is almost always

seen on the diaphragmatic boundary of the dextrogram, medial to the right costodiaphragmatic angle.

The right cardiac border joins with the right dome of the diaphragm at a sharp angle, the right cardiodiaphragmatic angle. Frequently this angle is bridged by a pale shadow which extends laterad and downward; this shadow, which is not invariably present, may first appear or may become more apparent with inspiratory descent of the diaphragm. The shadow is created by the right hepatic vein, for the upper edge of its orifice in the inferior vena cava usually projects above the diaphragm (Hasse, Elias and Feller, Hitzenberger). It should not be confused with adhesions which are common in the cardiodiaphragmatic angle; the vein can be distinguished easily since normally the median section of the diaphragm bends caudad without hindrance during inspiration whereas it is delayed and becomes stretched when it is fixed by adhesions in the cardiodiaphragmatic angle. Rarely, a similar shadow may be created by a collection of fat between the parietal leaf of the pericardium and the mediastinal pleura (Assmann, Herrnheiser, Kautz and Pinner).

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The left border of the mediastinal shadow has two strong convex arcs between which there is an indentation called the cardiac waist.

The cardiac waist varies greatly in depth This depends partly upon individual variations in the heart and great vessels and partly upon variable positional relations encountered by the heart in the thorax. Thus, elevation of the diaphragm may deepen the waist or make it shallow depending upon whether the heart becomes oblique or deformed by compression (p. 98); a slight right-convex scoliosis of the spine to the right (p. 252) may rotate the heart to the left while descent of the diaphragm with the heart, becoming perpendicular (p 100), may spread the waist.

The small arc often conspicuous above the cardiac waist and just below the clavicular shadows is the aortic knob. It is formed by the distal part of the aortic arch which has an almost sagittal course in contrast to the somewhat oblique path of the more proximal, pretracheal part, for this reason it has been called the "sagittal section" of the aortic arch (v. Jagić and Kreuzfuchs) (p. 365). The sagittal course of this part explains its relatively great density. Moreover, the knob corresponds to the left border of the aorta in the region of the isthmus. This is important in measurements of aortic diameter.

From the most lateral point of the aortic knob, the pale shadow of the descending aorta passes caudad, normally as a straight line converging slightly on the spine. Often a large part of it is visible in the cardiac waist and it can be followed downward for a variable distance in the cardiac shadow

From the upper edge of the aortic knob, the left border of the upper mediastinum rises obliquely in a lateral-concave direction, usually to vanish in the sternal end of the clavicle. Often it is seen to continue to the apex of the left lung, this is the shadow of the left subclavian artery (Assmann) Particularly in young people, as the edge of the shadow descends into the pulmonary arc (see below), it cuts across the aortic knob as if to remove a sector from it. This line indicates the course of the left anterior mediastinal pleura which descends on edge from the left subclavian artery to the pulmonary artery and is indented by the aortic arch which projects to the left.

The lowermost arc of the left edge of the mediastinal shadow is the left ventricular arc. It is formed by the anterior wall of the left ventricle which immediately adjoins the ventricular septum and belongs to the outflow tract of the left ventricle It is the longest and most rounded arc in the anterior image of the cardiovascular shadow. Its curve is somewhat parabolic (Schwarz) and its caudal end plunges into the abdominal shadow when the diaphragm is at a normal level or elevated To understand pathologic changes, one must remember the relation of the right ventricle to the left cardiac border. The anterior longitudinal sulcus which indicates the boundary between the left and right ventricles, runs close to, but somewhat steeper than, the left edge of the cardiac shadow. Near the base of the heart the right ventricle almost reaches the left cardiac border so that its outflow tract is separated from the left ventricle only by a short distance and it is separated from the left border of the heart by the small cupola of the left atrial appendage (see below). In many normal subjects it seems that the right ventricle with its pulmonary conus could form the border above the left ventricular arc.

Above the diaphragm the left ventricular arc turns sharply median to form the roentgenologic cardiac apex which usually is projected into the abdominal shadow.

Moritz and Dietlen believed that the cardiac apex could be outlined more often in recumbent patients than in erect ones We believe, however, that the recumbent position offers no advantage

in this respect although it must be conceded that when patients stand, the cardiac apex is often effaced by a large gastric gas bubble On the other hand, on lying, the heart sinks even more deeply into the diaphragm so that delineation of the apex can be more difficult than in upright patients. For easy demarcation of the apex, Huismans recommended the examination when fasting or after gastric lavage so that the diaphragm can descend as far as possible and the apex project freely. On the other hand, inflation of the stomach (Achelis) by an effervescent powder occasionally aids in outlining the apex but it may also have the opposite effect when the elevated diaphragm effaces it (Hammer). Apart from some instances of very large hearts and when a left hydrothorax exists, it is ordinarily possible, in our experience, to locate the cardiac apex. To facilitate this, the patient should take several deep breaths, this causes the apex to shift somewhat making it visible, since the eye is more sensitive to movement Palmieri advises a search in the right anterior oblique position for the place where the left lower cardiac border bends and to record it in the sagittal orthodiagram Thus, the site where the left cardiac border turns toward the diaphragm to form the apex should be ascertained more precisely by orthodiagraphy than by simple fluoroscopy in the sagittal position

When the heart is pendulous (fig. 55) the cardiac apex lies above the diaphragmatic shadow so that one can see between them, even the diaphragmatic surface of the heart, formed by the right ventricle, can be followed into the vertebral shadow and sometimes even to the right cardiac border.

In general, the roentgenologic cardiac apex is more rounded than in the cadaver heart Dietlen attributed this to the fact that in living subjects and in the postero-anterior position, the cardiac apex seems foreshortened owing to the oblique position of the heart, actually it may be more blunt than after death when this is accentuated by contraction and rigor mortis.

The anatomic cardiac apex is formed by the left ventricle. Whether this situation also prevails for the roentgenologic cardiac apex was the basis of a discussion between Dietlen and Zehbe The former believed that the right ventricle contributed as much if not more to the apex while the latter held that the left ventricle alone forms the roentgenologic apex, the right ventricle merely influencing its position The investigations of Arkussky as well as those of Laubry and coworkers with cadaver hearts filled with contrast agents and with angiocardiography in living subjects have actually shown that the roentgenologic cardiac apex of the anterior view normally is formed by the left ventricle, the level of which extends farther down and to the left than that of the right ventricle.

The palpable apical impulse corresponds to the roentgenologic apex in only one third of the cases (Dietlen), usually the former lies 1 to 2 cm. above and mesial to it (Schwarz, Dietlen, Sahli). This is explained by the fact that the cardiac apex which is superimposed by lung contributes less to the apical impulse than the anterior ventricular wall; the latter bulges during the period of contraction (systolic cardiac bulge) Only when the thorax is narrow may the apical impulse be located lateral to the left cardiac border (Dietlen). When cardiac action is excited by exertion, mental excitement, or thyrotoxicosis, the pulsation of the chest wall may extend far beyond the heart to the left despite a thorax of normal size (Moritz).

Ordinarily the diaphragmatic border of the heart cannot be distinguished against the abdominal shadow when the level of the diaphragm is normal, at times, however, a distinction can be made in a child or a slender adult with a pendulous heart. A flat, caudal-convex curve, drawn from the cardiac apex to the right cardiodiaphragmatic angle, approximates the course of the diaphragmatic border fairly well (Moritz). This can be convincingly demonstrated when the gas-filled colon happens

to be interposed between the liver and diaphragm (fig. 48) or a pneumoperitoneum (fig 14) allows one to recognize the lower limit of the central diaphragmatic tendon and consequently of the heart

The left cardiophrenic angle, like the right, is often filled by a pale shadow which descends laterad. Sometimes this shadow appears only in inspiration and when stretched by inspiratory descent of the diaphragm. It is straight or slightly concave but in deep inspiration its edge may also be laterally convex (fig 15) Occasionally it shows pulsations synchronous with, but smaller than, those of the left ventricle Schwarz called this shadow a "fat pad." It represents a collection of fat and con-

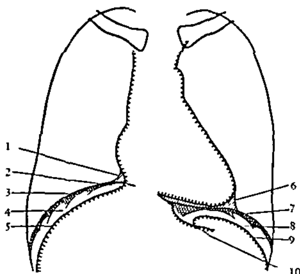


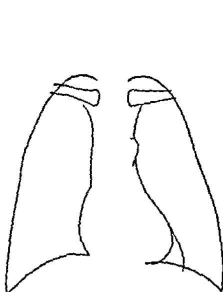
FIG 14 —Visibility of the diaphragmatic boundary of heart with pneumoperitoneum

1 Site of reflection of diaphragmatic pleura into the mediastinal pleura
2 V. hepatica dextra and cava inferior
3 Right diaphragm
4 Pneumoperitoneum
5. Liver
6 Fat pad
7 Left diaphragm
8. Pneumoperitoneum
9 Spleen
10 Lig coronarium hepatis

nective tissue between the diverging leaves of the parietal pericardium and mediastinal pleura (Assmann), usually it extends from the anterior chest wall to the velum of the phrenic nerve, a fold in the mediastinal pleura in which the phrenic nerve runs (Laurell). The fat pad is more common and often larger in obese individuals than in thin ones Possible confusion with adhesions in the cardiodiaphragmatic angle is easily avoided since the fat pad does not interfere with diaphragmatic movements during deep inspiration. If the border of the shadow is irregular and pleural adhesions are noted elsewhere on the left side, adhesions may actually be present (Pape). A physiologic fat pad can create difficulties in distinguishing the limit of the apex and cause errors in measurement of the longitudinal and transverse diameters of the heart unless a careful search with the shutter closely drawn is made to separate the more strongly pulsating, denser cardiac apex within the pale shadow Finally, a fat pad may at times simulate an aneurysm of the cardiac apex (p. 236) when its

edge is lateral-convex, this situation occurs, as we have already noted, not rarely in deep inspiration (Zdansky). Likewise atelectasis in the cardiophrenic angle occasionally assumes the shape of a fat pad (Pape)

Usually the cardiac waist can be resolved into two arcs The lower one tends to be so small and flat that its differentiation from the left ventricular arc is often uncertain It is formed by the left auricular appendage, which, to varying degrees in different individuals, here extends above the left ventricle from behind and even appears on the left cardiac border. If Laubry and coworkers never found a normal



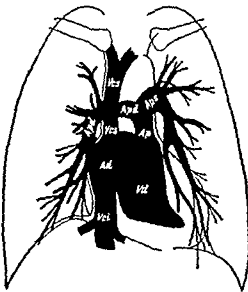


Fig. 15 Fig 16

Fig 15.—Fat pad with convex border Film in deep inspiration (copy of film).

Fig 16.—Position of right atrium and right ventricle, as well as the course of both Vv cavae and the pulmonary arteries (Schematic drawing after filling cadaver heart with contrast material, after Laubry, Cottenot, Routier, and Heim de Balsac) Vcs = superior vena cava, Vci = inferior vena cava, Ad = right atrium, Vd = right ventricle, Ap = pulmonary artery, Apd = right pulmonary artery, Aps = left pulmonary artery

left atrium, filled with contrast material after death, contributing to the contour, perhaps blood clots were present between the atrial trabeculae This may have prevented complete filling by opaque media and made roentgenologic demonstration of the auricular appendage impossible. As previously mentioned, the conus pulmonalis, which almost reaches the left border of the heart, sometimes actually [illegible]

the left border of the trunk of the pulmonary artery which arises from the conus craniad and posteriorly to divide into two main branches below the aortic arch The site of division is not demonstrable in the posteroanterior position It is projected almost into the upper end of the pulmonary arc.

to be interposed between the liver and diaphragm (fig. 48) or a pneumoperitoneum (fig. 14) allows one to recognize the lower limit of the central diaphragmatic tendon and consequently of the heart.

The left cardiophrenic angle, like the right, is often filled by a pale shadow which descends laterad. Sometimes this shadow appears only in inspiration and when stretched by inspiratory descent of the diaphragm. It is straight or slightly concave but in deep inspiration its edge may also be laterally convex (fig 15). Occasionally it shows pulsations synchronous with, but smaller than, those of the left ventricle. Schwarz called this shadow a "fat pad." It represents a collection of fat and con-

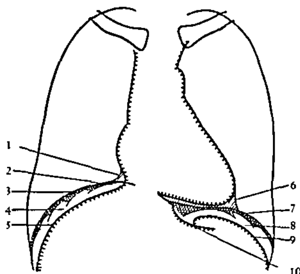


FIG 14 —Visibility of the diaphragmatic boundary of heart with pneumoperitoneum

1. Site of reflection of diaphragmatic pleura into the mediastinal pleura
2 V. hepatica dextra and cava inferior
3. Right diaphragm
4 Pneumoperitoneum
5. Liver
6 Fat pad
7. Left diaphragm
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9 Spleen
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nective tissue between the diverging leaves of the parietal pericardium and mediastinal pleura (Assmann), usually it extends from the anterior chest wall to the velum of the phrenic nerve, a fold in the mediastinal pleura in which the phrenic nerve runs (Laurell). The fat pad is more common and often larger in obese individuals than in thin ones. Possible confusion with adhesions in the cardiodiaphragmatic angle is easily avoided since the fat pad does not interfere with diaphragmatic movements during deep inspiration If the border of the shadow is irregular and pleural adhesions are noted elsewhere on the left side, adhesions may actually be present (Pape). A physiologic fat pad can create difficulties in distinguishing the limit of the apex and cause errors in measurement of the longitudinal and transverse diameters of the heart unless a careful search with the shutter closely drawn is made to separate the more strongly pulsating, denser cardiac apex within the pale shadow. Finally, a fat pad may at times simulate an aneurysm of the cardiac apex (p. 236) when its

the cardiac shadow; consequently they are not visible in most anterior views. The relations of the pulmonary veins are apparent in figure 17 which was drawn by Laubry and coworkers.

The view expressed long ago by Holzknecht has been confirmed, namely, the hilar shadows and "lung markings" owe their appearance, for practical purposes, to the vessels. Careful inspection of them is an essential part of every roentgen study

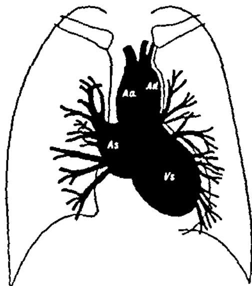


FIG 17.—Course of the pulmonary veins and position of the left atrium and left ventricle (Schematic drawing after filling cadaver heart with contrast material, after Laubry, Cottenot, Routier, and Heim de Balsac) As = left atrium, Vs = left ventricle, Aa = ascending aorta, Ad = descending aorta

of the heart because no other procedure affords a better insight into the blood and fluid content of the pulmonary circulation.

2. *The Right Anterior Oblique View in the Erect Position* (*Figure 19a and b*)

With rotation of the patient to the left as originally recommended by Holzknecht and von Criegern, the left atrium and ventricle progressively vanish while the right ventricle, approaching the anterior chest wall, progressively contributes more to the left border of the cardiac shadow With rotation of about 45 degrees (position 315) a large part of the left border is formed by the right ventricle, near the diaphragm, however, a section of the left ventricle is occasionally perceptible as a shallow curve. At the upper end of the right ventricular arc, the conus pulmonalis, merging with the beginning of the trunk of the pulmonary artery, protrudes like a flat knob.

The right atrium which formed most of the right border in the posteroanterior position is, by this time, projected largely into the cardiac shadow, but as a flat convex arc it still contributes extensively to the caudal right cardiac border. With increasing rotation to the left, however, it gradually disappears, replaced by the

The left branch of the pulmonary artery may be considered a direct continuation of the trunk (fig 16) It proceeds at first almost in the same plane, dorsad and cephalad, soon to bend over the root of the lung to the left, there it crosses the left main bronchus to pass over it at the posterior border of the upper lobe bronchus The angle of its slope, directed obliquely to the left, is subject to great individual variations In children it proceeds almost transversely (Kreuzfuchs), while in adults its course is more in a dorsoventral plane Consequently in children the left branch of the pulmonary artery often appears below the aortic knob like a comma, in adults, on the contrary, the left branch is often projected into the pulmonary trunk so that only its smaller branches are seen almost in the cardiac waist and perhaps even partly covered by it The oblique course of the left pulmonary artery corresponds almost exactly to that of the aortic arch to which it is bound by the short Lig Botalli (Kreuzfuchs) Naturally there are exceptions for an oblique course of the left pulmonary artery may be associated with a sagittal course of the distal aortic arch when the Lig Botalli is long

The right pulmonary artery is definitely longer than the left Below the arch of the aorta it bends to the right and continues almost horizontally in a transverse direction or descends a little behind the ascending aorta and superior vena cava to the root of the lung The right branch also is somewhat lower than the left Therefore it is somewhat below the right main and upper lobe bronchus whereas the left branch—as already mentioned—proceeds craniad to the left main bronchus to reach the posterior border of the upper lobe bronchus

Both branches of the pulmonary artery subdivide into superior and intermediate trunks just before entering the lung (Herrnheiser and Kubat) The superior trunks ascend to supply the upper lobes The intermediate trunk, as a direct continuation of the main branch, provides branches to the rest of the lung, it is located at the lateral border of the large bronchi which run downward

Assmann at first pointed out that the branches of the pulmonary artery form the major constituents of the hilar shadows which lie on each side of the cardiovascular shadow. The pulmonary veins, the bronchopulmonary lymph nodes and connective tissue have subordinate significance Angiocardiography in living subjects has confirmed this statement (Egas Moniz, Lopo de Carvalho and Almeida Lima, Lopo de Carvalho and Egas Moniz, Conte and Costa, Ravina, Robb and Steinberg).

Owing to the course of the two branches of the pulmonary artery, the left hilar shadow is somewhat higher than the right By and large both look like commas with their curves lateral and convex, both have branches above, below, and lateral, corresponding not only to the branches of the arteries but to the veins as well. Positive differentiation of the arterial from venous branches is possible only to a limited extent

On the right and left an upper and lower group of pulmonary veins are distinguished anatomically On both sides the main branches of the upper group pass in front of the arteries from the lateral and upper parts of the lungs toward the heart and then bend dorsad under the pulmonary artery toward the left atrium in which they empty Consequently the "antler-like" (Laubry) veins lie between the branches of the superior trunk of the pulmonary artery which ascend laterally and cannot be definitely distinguished from them The large veins are located at the lower border of the intermediate trunk and unite with them to form a band representing the union of the mediastinal and hilar shadows The large branches of the lower group of pulmonary veins are situated more caudad and behind the pulmonary artery. Their [illegible] in the middle and lower parts of the lungs and proceed in a med-[illegible]

border (Assmann) The large venous trunks of this group tend to be projected into

Holzknecht's space contains shadows of various posterior mediastinal structures, including the descending aorta, the esophagus, and lymph nodes. In the retrovasal space, almost parallel to the vertebral shadow, one sees the clear band of the trachea, a finger in width, soon divide into two branches. The clear band of the right main bronchus represents an almost straight continuation of the trachea; gradually it fades into the blurred shadows of the vessels of the right lung. The left main bronchus descends concavely and to the left almost parallel to the left side of the vascular band and finally fades into the darkness of the cardiovascular shadow, nevertheless the left upper lobe bronchus, rising obliquely, can usually be recognized.

The angulus of the left scapula is projected in the upper retrovasal space, in the middle part, single large branches of the right pulmonary vessels produce shadows; in the lower section some of the right breast shadow appears in women.

All these shadows blur the edge of the posterior cardiac wall and the vascular shadow against the posterior mediastinum more than one might anticipate. In muscular as well as obese individuals great difficulties arise. When the lungs are dark and hilar shadows are enlarged owing to pulmonary congestion or when a pleural effusion happens to exist, demarcation of the posterior cardiac wall may be impossible

Fortunately we possess three measures to facilitate visualization of the posterior cardiac wall even under adverse circumstances (1) The Frik maneuver, (2) Deep inspiration, and (3) Filling the esophagus with barium.

1. Frik's maneuver eliminates the disturbing shadow of the left scapula from the retrovasal space. The patient places his left hand on the occiput, or he may move his left forearm forward and lift it with right-angled flexion of the elbow so that the abducted upper arm is almost horizontal and is simultaneously rotated inward, then the shadow of the scapula moves out of the retrovasal space above the vascular band into the left lung field.

2. Inspiratory descent of the diaphragm, leads to elongation, the inspiratory lifting of the heart with the anterior chest wall, to widening of the posterior mediastinum, the retrocardiac space increases in length and breadth (fig 20). Since the lungs fill with air and the posterior mediastinum simultaneously broadens, transparency of the space often permits satisfactory visualization of the posterior cardiac wall even under adverse circumstances.

3. If difficulties still persist, it is advisable to visualize the position of the posterior cardiac wall indirectly by filling the esophagus with barium (fig. 21) (Schwarz, Assmann, Gäbert). The basis for success with this procedure is the close topographic connection between the heart and the esophagus. Only the upper thoracic esophagus lies directly in front of the spine, below the bifurcation it moves forward as the descending aorta, coming from the left, is interposed. Consequently, the esophagus steadily approaches the posterior cardiac wall until they are in contact for 2 to 4 cm If the esophagus is filled with barium, it is seen behind the heart, descending in a straight line or a slightly ventrad-concave curve; occasionally it shows a flat indentation (Conrads) and often a dorsally directed, transmitted systolic pulsation results from its close application to the posterior cardiac wall. If the latter encroaches unduly on the posterior mediastinum, perhaps from an enlarged left atrium, dorsal displacement of the esophagus (Assmann, Gabert) can be demon-

posterior wall of the left atrium. With rotation of about 60 degrees (position 300) this forms most of the right cardiac border except for a small supradiaphragmatic section (figs. 18, 19a and b, item 9). The special advantage of this position is the opportunity afforded to judge the size of the left atrium.

To the right and above, the cardiac shadow extends into the vascular band Below, it is broadly applied to and is indistinguishable from the abdominal shadow.

In this rotation to the left the spine is projected out of the cardiac shadow to the right and one may see between it and the heart. The clear field between them is

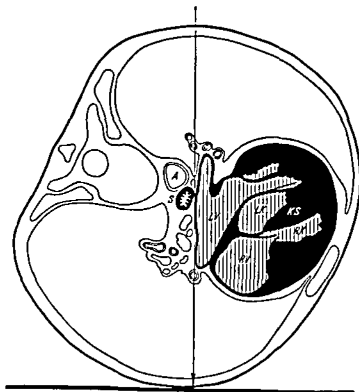


FIG 18 —With rotation to the left of about 60 degrees, the left atrium forms the border over a considerable area on the right side LV = left atrium, LK = left ventricle, RV = right atrium, *RK = right ventricle, KS = ventricular septum, S = esophagus, A = descending aorta* (Horizontal section through the thorax at the level of the eighth thoracic vertebra, after Pernkopf)

Holzknecht's space, the caudal section is called the retrocardiac and the cephalad portion the retrovasal space (Groedel and Arnsperger) This field is bounded on the right by the shadow of the spine, below by the diaphragm, and on the left by the cardiovascular shadow

The right border of the cardiac shadow is formed, as already mentioned, mainly by the left atrium, this describes a flat convex arc which caudad bends more noticeably to the left to join the diaphragm at a sharp angle. This angle is bridged by a pale shadow which rises obliquely and corresponds to the posterior border of the supradiaphragmatic section of the inferior vena cava. This vessel becomes more prominent with inspiratory descent of the diaphragm (fig 19b, item 11).

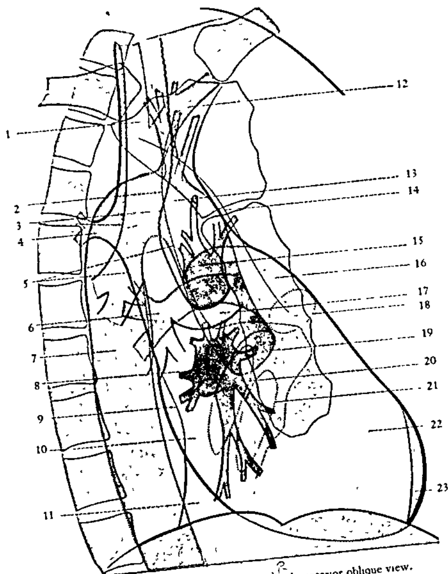


Fig 19b —Anatomic analysis of the right anterior oblique view.

1. V. brachiocephalica dext
2. Ant border of sup vena cava
3. Trachea
4. V thoracica longitudinalis dext (V. azygos)
5. Superior vena cava
6. Right pulmonary artery
7. Descending aorta
8. Left pulmonary veins
9. Left atrium
10. Right atrium
11. Inferior vena cava
12. V brachiocephalica sin
13. Aortic arch
14. Left mediastinal pleura
15. Left pulmonary artery
16. Trunk of pulmonary artery
17. Left upper lobe bronchus
18. Projection of pulmonary ostium
19. Projection of aortic ostium
20. Projection of tricuspid ostium
21. Projection of mitral ostium
22. Right ventricle
23. Left ventricle

strated (p. 162). Reference will be limited here to the fact that cardiac elevation by the diaphragm may make the posterior surface of the heart rounder and may lead to a circumscribed posterior displacement of the esophagus (fig. 22). This dorsal displacement can, however, be eliminated by abolishing diaphragmatic elevation with



Fig. 19a —Right anterior oblique view.

deep inspiration, then, as inspiration stretches the posterior cardiac surface, the esophagus also stretches.

Passing upward from the cardiac shadow is the vascular band, about one and one-half fingers in breadth and converging on the spinal shadow. Its demarcation requires special care and is facilitated by Frik's maneuver. In the formation of this

cephalic vessels (Frik). Sometimes a thin, sharply defined line spans the anterior edge of the vascular band; this line (fig. 19b, item 14) is formed by that portion of the left mediastinal pleura which is seen end-on proceeding to the left costomediastinal sinus (Zdansky). Since, in this region, variable amounts of fat are deposited between the great vessels and the pericardium on one side and the mediastinal pleura on the other, one may be able to see between the bandlike pleura and the mediastinal shadows. The presence of this pleural shadow and the breadth of the clear space joining it to the vascular band naturally depends upon the course of the pleura and the amount of mediastinal fat, consequently it is often missed or is perceptible only in very definite planes. If the mediastinal pleura is scarred, the line can be thick.

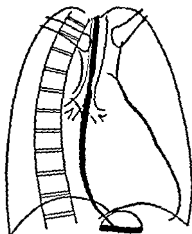


Fig. 21

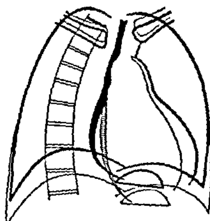


Fig 22

Fig. 21.—Normal course of esophagus in the right anterior oblique position.

Fig. 22.—Circumscribed posterior displacement of esophagus from cardiac elevation by high diaphragm (orthodiagram drawn with heavy line) Stretching of posterior cardiac wall and disappearance of outward esophageal bend in deep inspiration (orthodiagram drawn in thin line).

The right border of the vascular band, facing the retrovasal space is much more difficult to define than the left It is formed below by the superior vena cava, above by the right brachiocephalic (innominate) vein which turns to the right On films, this boundary is usually subdivided into two flat concave arcs, in juxtaposition just above the bifurcation (fig. 19a and b) At this place a short pointed appendage directed to the right indicates the orifice of the azygos vein in the superior vena cava (fig. 19b, item 4).

With less rotation the shadows of the superior vena cava and the right innominate vein project to the right of the clear tracheal band, with greater rotation, on the other hand, they are projected into this band (fig. 23). At all events, owing to the effacing effect of the trachea and adjoining left main bronchus, the vascular band is divided longitudinally into a right, narrower, light section and a left, broader, darker one (Frik). If the trachea produces a strong effacing effect, it may be difficult to recognize the posterior border of the vascular band With good films, however,

band, the ascending aorta, the pulmonary artery, and the superior vena cava participate. The descending aorta does not contribute in this position since it is projected toward the right with rotation of about 60 degrees, then its dorsal portion is located in the spinal shadow and the ventral in retrovasal space Deep inspiration defines it more clearly in the retrovasal space as a pale shadow projecting to the left of the spine (Frik).

The ascending aorta does not overlap the descending in this position although this almost happens with somewhat slighter rotation, perhaps at about 30 degrees.

With rotation to the left amounting to about 60 degrees, normally the aortic

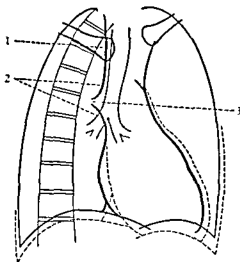


Fig 20—Widening and elongation of the retrocardiac space in deep inspiration (——— medium breathing, ------deep inspiration)

1 V. brachiocephalica dext (V anonyma dext)
2 V. cava superior
3. Orifice of azygos vein in the superior vena cava

arch can be defined only as far as the clear tracheal band (see below) which shines through it, providing the aorta is not abnormally wide or thick.

Near the bottom of the vascular band, just above the base of the heart, there is a round or oval shadow corresponding to the cross section of the left pulmonary artery where it bends over to the hilus (Frik). With good views the cross section of the vessels appears distinctly embedded in the angle formed by the upper lobe bronchus and the main bronchus (fig. 19b, item 15).

Below this section of the pulmonary artery but within the cardiac shadow there are one or more particularly dense shadows which emerge with offshoots on all sides: the left pulmonary veins and their branches (Assmann).

Above the flat bulge of the conus and the pulmonary artery, the left border of the vascular band proceeds at first for a short distance straight up in a direction convergent on the spine and then bends concavely to the left and outward. The short section, almost straight or slightly convex to the left, is the anterior wall of the aorta; the adjoining concave section, however, is formed by the left brachio-

taining where the distance seems smallest (de Abreu) Apart from the fact that this measurement, in a strict sense, no longer concerns the ascending aorta but is already in the domain of the aortic arch, there are certain difficulties in securing it. First, the point on the left vascular border located at the height of the left tracheobronchial angle need not correspond to one on the aorta exactly opposite the angle (Assmann), second, the left border of the vascular shadow at this level often is no longer formed by the aorta but rather by the trunks of the brachiocephalic vessels.

3. *The Left Anterior Oblique View in the Erect Position (Figure 25a and b)*

The left anterior oblique (boxing) position is employed almost exclusively for examining the aorta and both ventricles. With proper rotation of the patient to the

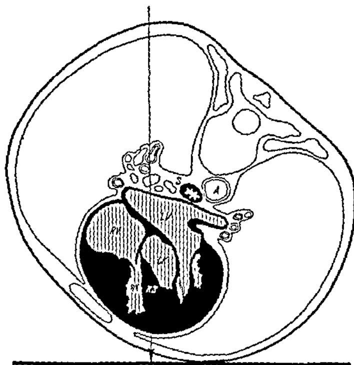


FIG 24 —With rotation to right to about 40 degrees, the ventricular septum courses approximately in the projection path of the ray so that both halves of the heart extend almost equally to left and right In this position, the left heart is partly projected into the vertebral shadow. LV = left atrium, LK = left ventricle, RV = right atrium, RK = right ventricle, KS = ventricular septum, S = esophagus, A = descending aorta (Horizontal section through the chest at the level of the eighth thoracic vertebra, after Pernkopf)

right, in this position, the ventricular septum follows the central beam of the x-ray in such a way that the two halves of the heart are divided about equally to right and left and their sizes can be compared (fig. 24) (O'Kane, Andrew and Warren, Nemet and Schwedel, Fray).

Naturally the ventricular septum itself always remains invisible so that we possess no *proof* that at a certain angle of rotation it actually proceeds exactly in the path of the ray The angle which

this can usually be accomplished by following Frik's suggestion: seek the uppermost end of the right border of the vascular band; this is formed by the right innominate vein at the level of the right clavicle, use this as a guide and follow it downward (fig. 19a and b)

Lippmann and Quiring as well as Vaquez and Bordet mistakenly considered the right, partly effaced portion of the vascular band as a part of the superior vena cava which projects beyond the shadow of the ascending aorta posteriorly and to the right. Consequently they regarded the junction between the light and dark parts of the vascular band as the posterior wall of the aorta and accordingly based their measurement of the diameter of the ascending aorta in the right anterior oblique position on a rotation of 30 to 40 degrees

From this discussion it is evident that the darker part of the vascular band opposite the spine is not the aorta but the section of the band which is not effaced

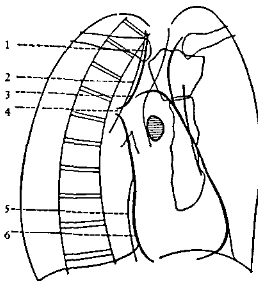


FIG 23 —With greater rotation to the left, the right innominate vein and the posterior wall of the superior vena cava can be projected into the clear band of the trachea

1 V anonyma dext
2. Posterior wall of trachea
3 Posterior boundary of V cava sup
4 Orifice of V azygos in V cava sup
5. Ant boundary of descending aorta
6 Posterior wall of heart

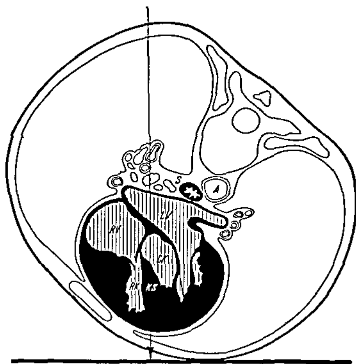
by the clear trachea and the left main bronchus. On the whole, the right posterior border of the ascending aorta is not exactly perceptible unless the vessel is pathologically dilated or thick. There is also no guarantee that the section of superior vena cava which projects beyond the aorta corresponds precisely to the portion of the band which is effaced by the trachea and the left main bronchus. Therefore, exact measurement of the diameter of the ascending aorta in the right anterior oblique position is not possible at any angle of rotation (p 363).

According to Assmann and de Abreu the diameter of the aorta is most nearly obtained by making the measurement higher, the distance from the left tracheobronchial angle, which is usually easy to see, to the anterior border of the vascular band is used (fig 260); the degree of rotation of the patient is determined by ascer-

taining where the distance seems smallest (de Abreu). Apart from the fact that this measurement, in a strict sense, no longer concerns the ascending aorta but is already in the domain of the aortic arch, there are certain difficulties in securing it. First, the point on the left vascular border located at the height of the left tracheobronchial angle need not correspond to one on the aorta exactly opposite the angle (Assmann), second, the left border of the vascular shadow at this level often is no longer formed by the aorta but rather by the trunks of the brachiocephalic vessels.

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atrium, LK = left ventricle, RV = right atrium, RK = right ventricle, KS = ventricular septum, S = esophagus, A = descending aorta (Horizontal section through the chest at the level of the eighth thoracic vertebra, after Pernkopf)

right, in this position, the ventricular septum follows the central beam of the x-ray in such a way that the two halves of the heart are divided about equally to right and left and their sizes can be compared (fig 24) (O'Kane, Andrew and Warren, Nemet and Schwedel, Fray).

Naturally the ventricular septum itself always remains invisible so that we possess no proof that at a certain angle of rotation it actually proceeds exactly in the path of the ray. The angle which

it forms with the frontal plane varies from case to case. It changes with the position of the heart in the thoracic cage and with the size of the two ventricles, a transverse position of the heart from an elevated diaphragm is connected with a rotation to the left, a perpendicular position by virtue of diaphragmatic descent rotates the heart to the right with resultant change in the septum. Assmann first drew attention to rotation of the heart to the left as the result of right ventricular hypertrophy (p 143) Later Kirch and his coworkers succeeded in analyzing more exactly the cardiac rotation resulting from alterations of the chambers They found that widening of the outflow tract of the right ventricle led to a rotation of the heart to the left while dilatation of the inflow tract opposed this rotation and might even reverse it In animal experiments Döring found an analogous situation for the left ventricle a rotation of the heart to the right with widening of the outflow tract of the left ventricle diminished or completely disappeared with predominant dilatation of the inflow tract. All these conditions, partly intracardiac and partly extracardiac, may also alter the angle which the ventricular septum forms with the frontal plane in different ways, accordingly we possess no roentgenologic procedure for accurately determining the orientation of the ventricular septum nor can this be secured with reconstructions (O'Kane, Andrew and Warren, Fray). Such reconstructions imply a nonexistent exactitude, for the ventricular septum does not proceed in a single plane but is more or less bent

For practical purposes, however, one may assume that rotation of the patient about 40 degrees to the right brings the septum approximately into the path of the ray This subject will be discussed in greater detail in connection with pathologic alterations of the single cardiac chambers.

To examine in the left anterior oblique position at first the patient is rotated about 40 degrees to the right (Position 40) (fig. 25a and b). The right ventricle, which did not contribute to the right border of the cardiac shadow at all or formed only a short section as a shallow notch just above the cardiodiaphragmatic angle in the anterior view, now becomes increasingly apparent above the diaphragm, on the other hand, the right atrium and its appendix now participate only in the cranial portion of the right border The junction between the right atrium and ventricle is often visible as a shallow notch or indentation which corresponds to the coronary sulcus Often this is apparent only during deep inspiration when the atrium becomes rounder owing to the inspiratory influx of blood Usually the contours of the right atrium and ventricle are combined into a flat convex arc which inclines obliquely to the diaphragm In women this is often superimposed to a considerable extent by the anterior border of the left breast, making demarcation difficult.

On the left border of the cardiac shadow the left atrium forms an increasingly large upper section until, with rotation to the right of about 40 degrees it constitutes about the upper half, the lower half is formed by the left ventricle. Occasionally a little notch or indentation marks the atrioventricular junction The arc of the left cardiac border becomes apparent below the bifurcation in the retrocardiac space as the bifurcation emerges out of the maze of pulmonary vessel shadows; then it descends fairly obliquely and convexly to the left, to bend sharply to the right above the left diaphragm. Its lower end forms a pointed angle with the diaphragm and the vertex of this angle normally lies a few centimeters to the right of an imaginary perpendicular dropped from its upper end

The left border of the cardiac shadow is much rounder than the right. Both borders descend together and vanish in the abdominal shadow. With deep inspiration and descent of the diaphragm, the bridge joining the cardiac and abdominal shadow becomes increasingly narrower. In ptotic patients the cardiac shadow may

be lifted completely away from the abdominal; then one sees the two cardiac borders join in an ovoid globe (fig. 25). The sole connection now existing between the cardiac and abdominal shadows is the inferior vena cava whose shadow is approximately the width of a thumb. Somewhat to the left of the rounded lower pole of the cardiac shadow, the left and right ventricles join in the region of the cardiac apex. The approximate position of the apex in patients whose cardiac shadow sinks to a considerable extent into the abdominal shadow, may be assumed to lie where the lines meet when the courses of the two cardiac borders are prolonged.

In this position naturally the heart appears more or less shortened according to its angle of inclination If the heart is transverse, owing to a high diaphragm, the organ seems globular; if its position is perpendicular owing to diaphragmatic descent, an oval or pear shape is assumed

With this rotation the spine is projected out of the cardiovascular shadow to the left although the left border of the heart tends to be projected into the vertebral shadow with a flat segment cut off; then the retrocardiac field is bisected into a large area at the level of the bifurcation and a smaller clear triangle above the diaphragm. Only with small and median placed hearts or with very deep inspiration are the cardiac and spinal shadows entirely separate.

With further rotation to the right the cardiovascular shadow moves farther out of the spinal shadow and the retrocardiac field becomes progressively wider. At a rotation of 60 degrees or more the left atrium forms more and more of the border above the ventricle. Now, in the atrial domain strikingly large systolic pulsations are flung dorsad, and gradually merge below with the powerful, steady, systolic, ventrally directed pulsations of the ventricle, the two types alternate with each other The flapping pulsations belong to the left atrium and result from its brisk filling starting with ventricular systole (Zdansky and Ellinger).

The sharp angle formed by the left cardiac border and the diaphragm is filled by one, two, or three oblique shadows which may be followed upward for a variable distance into the cardiac shadow (figs 26–28). Their individual anatomic basis is uncertain and their position and number vary individually according to the angle of rotation and the level of the diaphragm In thin individuals with a low diaphragm and with deep inspiration, the line located farthest to the right, without doubt, is often the posterior wall of the inferior vena cava (fig. 26), this can be demonstrated convincingly by the induction of pneumoperitoneum Occasionally the vein can be followed upward toward the vascular band to provide a good idea of the course of the caval axis. If diaphragmatic position is normal, however, the posterior caval wall is not visible at this place. The anatomic basis for the other shadow or shadows in the cardiodiaphragmatic angle is uncertain. Presumably the folds of reflection of the mediastinal pleura and perhaps the Lig pulmonalia also play a role

In the angle between the right cardiac border and the diaphragm usually a linear shadow ascends toward the heart Sometimes this corresponds to the right anterior border of the inferior vena cava when one can see between the heart and the diaphragm owing to the low position of the latter. But when the cardiac shadow dips into the abdominal shadow, as is usually the case, the reflection of the diaphragmatic pleura into the mediastinal produces the shadow Often this can be followed up into the cardiac shadow from which it cuts off a small ventral segment of less density

(fig 26a and b). This clear ventral zone is produced by the air-containing lung interposed between the heart and the right anterior chest wall in the right costomediastinal angle Cephalad, the shadow edge merges directly with the right border of the vascular band or proceeds upward just within its right edge. Often it con-

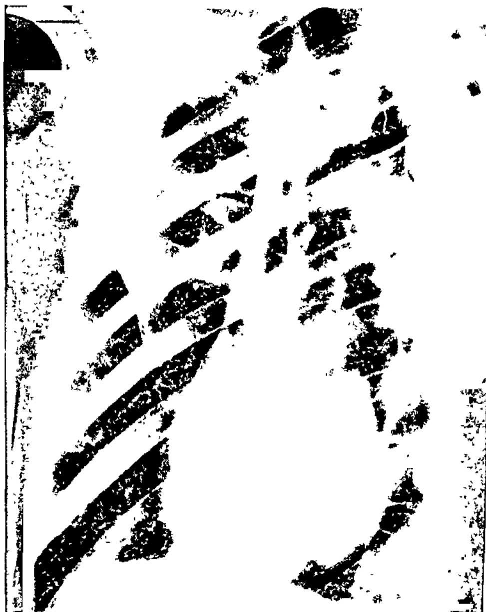


Fig 25a —Left anterior oblique view.

tinues as a linear streak bridging the right border of the vascular shadow which executes a gentle concave curve to the right (Zdansky). This shadow corresponds to a part of the right ventral mediastinal pleura which is seen on-end Its course depends upon the development of the right costomediastinal angle, which varies,

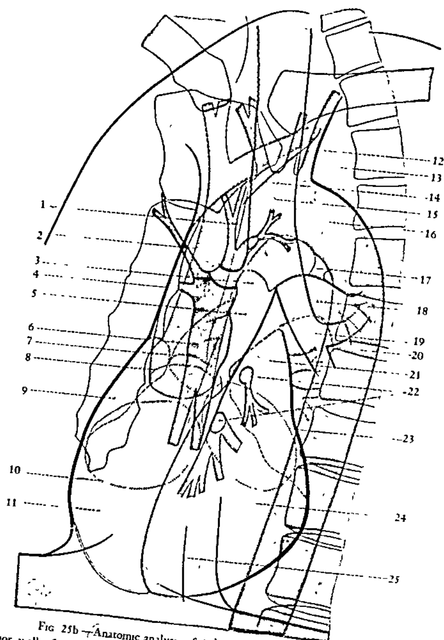


FIG 25b — Anatomic analysis of the left anterior oblique view.
1 Posterior wall of sup vena cava 2 Anterior wall of sup vena cava 3 Right pulmonary artery 4 Anterior wall of ascending aorta 5 Right bronchus 6 Right hilus 7 Projection of pulmonary ostium 8 Projection of aortic ostium 9 Right atrium. 10 Projection of tricuspid ostium 11 Right ventricle 12 Left subclavian artery 13 "Aortic triangle" 14 Transition of left costal into mediastinal pleura, end-on 15 Trachea 16 Aortic arch 17 Lig arteriosum 18 Left pulmonary artery 19 Left bronchus 20 Descending aorta 21 Left atrium 22. Right pulmonary veins 23 Projection of mitral ostium 24 Left ventricle. 25 Posterior wall of V cava inf

and naturally changes with the angle of rotation. At the level of junction of manubrium and corpus sterni, the stripe continues in an S-shape which is visible to the left above through the clear tracheal band as far as the end of the left clavicle (fig. 26a and b). This line indicates the transition of the left costal pleura, receding from the anterior chest wall, into the mediastinal pleura. The idea that this line is the direct continuation of the right ventral mediastinal pleura depends upon the close approach of both pleural cavities to each other near the retrosternal "anterior weak area" (Brauer) where they are almost in contact behind the manubrium



FIG 26a —Left anterior oblique position Rotation to right about 40 degrees

Naturally the shadows show considerable differences from case to case since the extent of each pleural space and the sites of costomediastinal reflection normally vary greatly, further complexities are created by acquired alterations of the pleura and lungs The possibility of demonstrating various pleural shadows depends, to a high degree, upon the plane of projection (figs 26 to 28) Consequently some of the shadows described are not perceptible in a given case

The dark vascular band rises obliquely craniad from the cardiac shadow (fig. 25a and b). It is composed primarily of the ascending aorta, the superior vena cava, and the right pulmonary artery Its breadth and definition vary greatly with the degree of rotation (figs. 26–28) Even slight rotation out of the dorsoventral passage of the ray suffices to hide the superior vena cava behind the broader aorta (Reich) The right border of the vascular band is formed by the section of ascending

aorta which lies directly above the right edge of the cardiac shadow and rises obliquely as a straight line or as a curve, slightly concave to the right, converging on the vertebral shadow. Farther cephalad the right vascular band bends concavely to the right through the sternal shadow toward the medial end of the right clavicle; the basis of this part is the innominate vein.

The left boundary of the vascular band is formed by the clear tracheal band, a

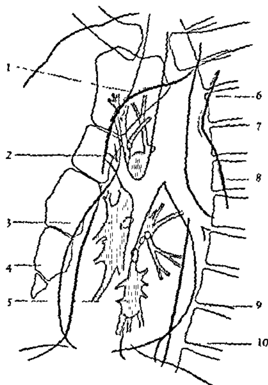


FIG. 26b.—Left anterior oblique position. Rotation to right about 40 degrees

1 On-end transition of the left costal pleura into the mediastinal pleura
2 Left mediastinal pleura
3. Right hilus
4 Right mediastinal pleura
5 Right pulmonary veins
6. Left subclavian artery
7. "Aortic triangle"
8. Posterior border of the descending aorta
9 Anterior border of the descending aorta
10. Inferior vena cava

finger in width, and the right main bronchus. The clear tracheal band descends from the neck at the right of the spine, nearly perpendicularly or curved slightly convex to the right; on the way it lights up all vascular shadows (see below). Near the level of the fifth thoracic vertebra it divides into two clear bands, the main bronchi. In most cases the bifurcation is readily seen. The right bronchus is an almost straight continuation of the trachea and is the actual left boundary of the vascular band, it can be followed to a variable depth in the cardiac shadow. Occasionally, in some positions and with certain planes of projection which vary from case to case, the clear band of the right upper lobe bronchus branches at a sharp angle to the right

above and cuts across the dark vascular band (figs. 26 and 28). The tapering righ lower lobe bronchus usually slopes more or less obliquely to the right a few centi meters below the bifurcation, to end suddenly at an acute angle within the cardia shadow (figs. 26 and 28). The slope and tapering are brought about by an intens oblong shadow, the upper pole of which extends to the vascular band, the lower to the cardiac shadow and then fades on all sides into branches. In the center of thi shadow, with a rotation of 40 to 60 degrees, an oval dark nucleus may project into the clear bronchial band to produce its slope This nucleus is the right pulmonary



FIG 27a —Left anterior oblique position Rotation to right about 50 degrees The width of the vascular band in this position corresponds to the ascending aorta

artery and its branches at the right hilus. Consequently, Frik is undoubtedly correct in stating that the left limit of the vascular shadow against the right bronchial band is normally formed by the right pulmonary artery and not by the ascending aorta, contrary to the statement of Reich (figs 25 and 26) In addition, the section of the right cardiac border opposite this slope, where one measures the aortic diameter in Reich's procedure is not usually the aorta at all but the right atrium, obviously measurement of the diameter of the ascending aorta at this level is not free from objections.

These remarks are not intended to imply that this diameter cannot be determined at this place under some circumstances They are valid, in general, only when the aorta is normal. With a wide and consequently dense aortic shadow the slope of

the right bronchus is actually formed by the posterior wall of the aorta and the opposite place on the right cardiac border by the anterior aortic wall, then the diameter can actually be measured at this level.

Under all circumstances, however, the conditions are more favorable in the portion of the ascending aorta which projects just above the right pulmonary artery. It is true that the width of the vascular band changes considerably at this level

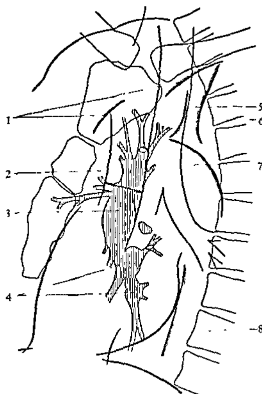


FIG 27b—Left anterior oblique position Rotation to right about 50 degrees The width of the vascular band in this position corresponds to the ascending aorta

1. On-end transition of the left costal pleura into the mediastinal pleura
2 Anterior border of ascending aorta
3. Right hilus
4. Right pulmonary veins
5. Art carotis communis sin. and Art. subclavia sin
6 "Aortic triangle"
7. Aortic arch
8 Anterior border of descending aorta

depending upon the amount of rotation to the right (figs. 26 to 28) The less the rotation is, the smaller the vascular band becomes since a part is progressively effaced by the clear trachea (fig. 26), with further rotation to the right, the band widens as the ascending aorta is detached more and more to the right from the trachea (fig 28), finally it separates completely. Moreover, it should be clear that the width of the vascular band at this place corresponds to the aortic diameter only when its shadow at this place just touches the light tracheal band This happens

only at a definite angle of rotation which varies individually and must be determined in each case. *To find this, it is best to turn the patient slowly to the right until the* vascular band is at the level of the tracheobronchial angle or just beneath it, with parallel edges on both sides (figs. 27 and 261), and stands sharply against the clear trachea This locates a plane where the shadow of the ascending aorta and the clear band of the right bronchus are no longer projected into each other but together so that they just touch.

It must be acknowledged that demarcation of the posterior wall of the ascending aorta at this level may encounter overwhelming difficulties owing to powerful



Fig. 28a.—Left anterior oblique position Rotation to right about 60 degrees.

muscular development, obesity, pulmonary congestion, pleural scars, or pulmonary fibrosis, in thin individuals and emphysematous patients, the lungs may be so radiolucent that they shine through parts of the aorta precluding definite delineation Even under otherwise normal conditions, however, the demarcation is hardly reliable as one will realize by recalling that the contrast of the ascending aorta against the right main bronchus does not depend upon their close relations but is subject to haphazard summations and subtractions of shadows of different densities. Moreover, the right border of the vascular band at this level often no longer corresponds to the anterior wall of the ascending aorta but to the innominate vein, this is evident since the edge of the shadow no longer parallels the posterior wall of the ascending aorta but extends to the right and concavely toward the sternal end of the right clavicle.

The proximal division and vertex of the aortic arch are not conclusively demonstrable. The first forms a mass of shadows with the right brachiocephalic vessels passing toward the neck, the latter is traversed by the clear tracheal band which shines through it so that its upper boundary is perceptible only at times as a pale cranioconvex curve With greater regularity the upper border of the distal aortic arch can be demarcated beyond the trachea. Below it completes a translucent tri-

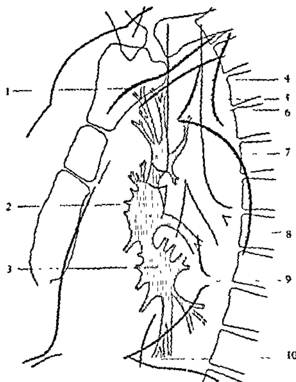


Fig 28b —Left anterior oblique position. Rotation to right about 60 degrees.

1 On-end transition of the left costal into the mediastinal pleura
2. Right hilus
3. Right pulmonary veins
4. Mammary fold (?)
5. Art carotis communis sin and Art subclavia sin.
6. "Aortic triangle"
7. Aortic arch
8 Posterior border of the descending aorta
9 Left pulmonary vein
10 Vena cava inferior

angle, the left side of which is the spine, and the right a flat shadow, concave to the left, descending obliquely from the left above (figs 25 to 28). The latter corresponds to the subclavian fold of the left mediastinal pleura in which the left subclavian artery runs This triangle, the breadth of which varies according to the amount of rotation, was named the "aortic triangle" by Parkinson and Bedford It corresponds to a sinus of the left pleural cavity toward the mediastinum which extends in front of the spine and behind the brachiocephalic vessels, air passages, and esophagus almost to the midline (fig 13) Here it almost contacts the corresponding pouch into the right pleural cavity (Danelius) The transparency of this

only at a definite angle of rotation which varies individually and must be determined in each case. To find this, it is best to turn the patient slowly to the right until the vascular band is at the level of the tracheobronchial angle or just beneath it, with parallel edges on both sides (figs 27 and 261), and stands sharply against the clear trachea. This locates a plane where the shadow of the ascending aorta and the clear band of the right bronchus are no longer projected into each other but together so that they just touch

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FIG. 28a.—Left anterior oblique position Rotation to right about 60 degrees.

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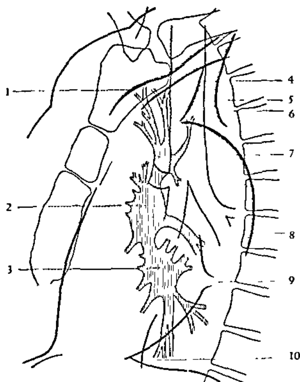


FIG 28b —Left anterior oblique position Rotation to right about 60 degrees

1 On-end transition of the left costal into the mediastinal pleura
2. Right hilus
3. Right pulmonary veins
4. Mammary fold (?)
5. Art. carotis communis sin. and Art subclavia sin.
6. "Aortic triangle"
7 Aortic arch
8 Posterior border of the descending aorta
9 Left pulmonary vein
10. Vena cava inferior

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triangle, so striking to the eye, is easily explained; at this place the posterior mediastinum represents only a reduplication of the pleura separated by a little connective tissue, the "dorsal mesentery" of the esophagus (see p. 20) (Pratje).

Sometimes when the rays pass in a certain plane a second shadow runs in a craniocaudal direction in the clear tracheal band It also parallels the subclavian shadow mentioned above but rests on the aortic arch with a flat curve, concave to the right It indicates the course of the left common carotid artery (figs 27 and 28).

The contours of the last two branches of the aortic arch mentioned above create a biconcave dark band which merges with the shadow of the aortic arch (Zdansky).

In some films a linear shadow begins below the left clavicle to cut downward across the trachea to the right and extend toward the left border of the manubrium (fig 28). This line corresponds to the mammary fold of the mediastinal pleura in which the left Vasa mammaria runs (Zdansky).

The inner border of the aortic arch cannot be defined unless the aorta is pathologically wide or its wall is calcified. Therefore, measurement of the aortic diameter is not normally possible in this region, contrary to the suggestion of Parkinson and Bedford Naturally the conditions are different when the aorta is dilated or calcified

The descending aorta can be outlined to some extent, but this differs individually and depends to a considerable degree on the angle of rotation By shadow analysis one may differentiate the ascending aorta within the spine as a darker shadow flatly curved to the left (fig. 28). Much more rarely the ventral border is demonstrable as a pale shadow located at the right of the spine with its free right border usually just below the clear left bronchus (figs 26 and 28) If parts of the anterior and posterior walls, lying opposite each other, are visible, it is possible to determine the diameter of the descending aorta, this, however, is rare with a normal aorta

It should be evident from this discussion that in the left anterior oblique view of the aortic loop, one may recognize the anterior wall of the ascending portion, the upper limit of the *distal portion of the arch, and parts of the posterior wall of the* descending vessel with considerable regularity On the other hand, the inner wall of the aortic loop normally can be defined, at most, at the level of the right tracheobronchial angle and sometimes in the region of the descending aorta The fact that the outer border of the aorta can be demonstrated relatively well has great value in roentgenologic evaluation for it gives a good idea of its course, span and the curvature of its divisions.

Normally the trunk of the pulmonary artery cannot be seen The left pulmonary artery, which proceeds dorsad and almost sagittal to bend over the left main bronchus to the root of the lung (p 25), is frequently visible In thin individuals and particularly when the vessel is dilated it can be recognized as a band, a finger in width, arching upward to the left, to cross over the clear left main bronchus below the aortic arch and finally to fade into the vertebral shadow (figs. 25 and 300)

Occasionally one can recognize the Ligamentum arteriosum *Botalli* bridging the shadows of the aortic arch and the left pulmonary artery (fig 330)

Usually the initial portion of the right pulmonary artery cannot be outlined although its division in the root of the right lung ordinarily is easily seen. The latter, as mentioned above, is projected into the left portion of the vascular band to form its

left border where the vascular band passes out of the cardiac shadow. With a rotation of about 30 to 45 degrees the artery appears as an oblong shadow which shows branches on all sides but particularly above and below (figs. 25 to 28). Here the right hilar shadow confronts us, shining through the cardiovascular shadows. With a rotation of 50 to 60 degrees, a dark, round or oval center can often be recognized within the hilar shadow, corresponding to the cross section of the right pulmonary artery immediately before its division into large branches.

In the center of the cardiac shadow itself, directly below the area in which the right pulmonary artery subdivides and usually combined with it, one or two dark, round or oblong areas from which dendritic projections extend in all directions may be seen (figs. 25 to 28). These are the right pulmonary veins which—usually divided into two groups—empty into the atrium (Assmann).

Of the left pulmonary veins only a few branches directed to the left are visible.

4 *The Left Lateral View in the Erect Position (Figure 29)*

With transverse dextro-sinistral projection the degree of inclination of the heart from the back-above to the front-below is distinct.

Between the heart and the anterior chest wall one sees the clear retrosternal field corresponding to the anterior mediastinum It has the shape of a small triangle with the apex downward. Below the attachment of the fourth rib, approximately, the cardiac shadow no longer can be distinguished from the sternum. Often, however, just above the diaphragm, the heart again retreats somewhat so that a second smaller clear triangle develops; its apex is directed upward.

Palmieri and Rösler emphasized that to the left of the midline the heart may project ventrad beyond the posterior surface of the sternum when it is situated in the curvature of the left anterior chest wall Since in a lateral view some of the anterior cardiac wall may be projected into the shadow of the sternum (fig 29) it may not be correct, without further consideration, to locate the anterior heart surface on the posterior aspect of the sternum

In inspiration the retrosternal field becomes broader and lighter since the heart follows inspiratory elevation of the anterior chest wall imperfectly. Consequently the chest wall may separate from the heart enough for one to see between them Since the retrosternal field often fails to expand, this fact alone does not mean that local adhesions exist The demarcation of the retrosternal field is often difficult in women since the breast shadows are projected into it. In patients with emphysema the field is usually wide and may even extend to the diaphragm

The right border of the cardiac shadow is formed by the flat, convex arc of the anterior right ventricular wall to which the flat bulge of the conus and the pulmonary artery is joined above Then the anterior wall of the ascending aorta ascends to the left almost in a straight line

Between the cardiac and vertebral shadows is the retrocardiac space, normally it is rather small and dark but greater breadth and radiolucency is attained in ptotic individuals and patients with emphysema. In young patients, demarcation of the cardiac shadow from the posterior mediastinum usually offers no problem, in

stout and muscular individuals, however, and with diseases of the heart and lungs, distinction is often impossible.

The left border of the cardiac shadow is formed largely by the left atrium (Assmann, Gabert). The left ventricle may participate in this border but only in the lowermost section and at a sharp angle where the border joins the diaphragm which descends posteriorly Since the angle is regularly bridged by the pale, obliquely rising supradiaphragmatic section of the inferior vena cava (usually curved slightly concave to the left) the left ventricle does not normally form this border with frontal

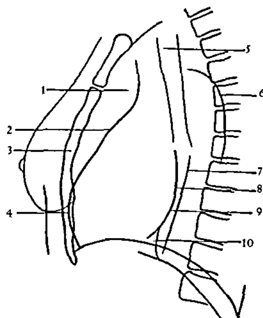


FIG. 29 —Left lateral view.

1. Retrosternal field
2. Pulmonary artery
3. Sternum
4. Right ventricle
5. Trachea
6. Aortic arch
7. Anterior wall of descending aorta
8. Retrocardiac field
9. Left atrium
10. Inferior vena cava

passage of the ray (Gäbert) Only when this chamber is dilated or hypertrophied, in deepest inspiration or with marked emphysema, does the left ventricle appear above the shadow of the inferior vena cava (Assmann).

The left lateral view is employed primarily to determine the antero-posterior diameter of the heart and to investigate the anterior mediastinum

5. The Right Lateral, the Posterior, and the Right and Left Posterior Oblique Views in the Erect Position

Examinations in these positions reveal only slight differences from those with opposite projection. The trifling deviations are merely the result of central pro-

jection With orthodiagraphic projection identical pictures are secured. Consequently, no special discussion of these positions seems necessary.

On the other hand, investigations of the cardiovascular complex in the dorsal recumbent position as well as in the right and left lateral recumbent positions with dorsoventral projection is important.

6. The Anterior View of the Heart in the Dorsal Recumbent Position

Apart from an increase in size (p 90ff) the cardiac shape changes its configuration in the dorsal recumbent position (Moritz). This transformation (fig. 30) is brought about mainly by altered spatial relations. Elevation of the diaphragm shortens the thoracic cavity and pushes the heart upward. Owing to its oblique axis and

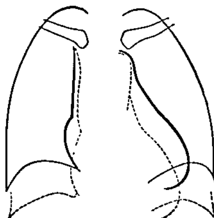


FIG 30 —Alteration of cardiovascular shadow by change from erect to dorsal recumbent position. (------Erect position, ———Dorsal recumbent position.)

the relative fixation between the two venae cavae, the heart becomes oblique, whereby the left cardiac border tends to bend laterad somewhat more than the right Elevation of the pulmonary artery makes the cardiac waist less differentiated and more curved. Sometimes the pulmonary arc also bulges. The old debate as to whether the cardiac apex is defined better in the recumbent patient than in an erect one cannot be settled for all cases Certainly demarcation is more exact in the recumbent position than when a large gas bubble obscures the apex of the erect patient Apart from this, examination with the patient recumbent offers no advantage in this respect

The aortic knob appears a little higher and approaches the clavicular shadow Consequently the vascular band is shorter and broader since both borders bend laterad, the superior vena caval shadow in particular appears more clearly and usually rises in a straight line

The poorer arrangement of the cardiac shadow resulting from the darker lung fields plus the difficulty of rotating the patient in the recumbent position make it less suitable for the analysis of the cardiac shadow than the erect posture. On the other hand it is more satisfactory for determinations of cardiac size.

7. *The Anterior View of the Heart in the Right and Left Lateral Recumbent Positions*

The cardiac shadow experiences very striking alterations in the right and left lateral recumbent positions. These changes involving position as well as shape are essentially effects of gravity. Owing to gravity the mediastinum sinks to the side on which the patient reclines. Consequently the diaphragm on this side ascends into

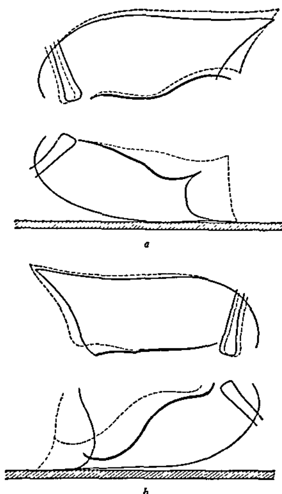


Fig. 31.—Respiratory wandering of mediastinum and alterations of cardiac shape from respiration. *(a) Right lateral position, (b) left lateral position (——— average breathing, ------ deep inspiration.)*

the thoracic cavity while the dome on the upper side descends and flattens (fig. 31a and b).

The descent and flattening of the upper diaphragmatic dome is produced by the reduction of intra-abdominal pressure on this side, conditioned by gravity, and by the pull exerted by the heart sinking into the lower side; the elevation of the lower dome of the diaphragm is explained by the augmented intra-abdominal pressure on this half and to relaxation resulting from descent of the heart.

The alterations of diaphragmatic support naturally assist in transforming the shape of the heart, the nature and extent of which is influenced by the suspensory and fixation apparatus of the heart and by cardiac plasticity, this varies individually.

In the right lateral position (fig. 31a) the heart as a whole sinks to the right and in addition it rotates like a "swinging door" (Determann) around the caval axis to the right. This displacement makes the left cardiac border more tense so that the cardiac waist becomes more shallow while the right cardiac border is pushed up by the elevated right dome of the diaphragm and projects more roundly into the lung field

In the left lateral position (fig 31b) the apex of the heart sinks to the left and rotates to the right from elevation of the left diaphragm, the heart is lifted so that its waist deepens and its left border projects farther into the left lung field in keeping with a lateral shift of the entire heart The right cardiac border, lying near the caval axis, alters its position and shape relatively little in comparison to these features in the erect position, and is projected largely into the vertebral shadow, the result is merely a slight flattening of the arc of the right atrium and often greater exposure of the right hepatic vein in the right cardiodiaphragmatic angle.

The extent of lateral cardiac shift varies decidedly in different individuals On the basis of extensive studies with percussion and palpation Determann concluded that, generally speaking, cardiac mobility is greater in women than in men, in young children less than in older ones and adults, and that it again decreases at more advanced ages. He found the extent of mobility determined by the stability of the connective tissue and vascular suspensory apparatus, by spatial intrathoracic conditions, and by pulmonary and diaphragmatic resistance. A pliant suspensory apparatus, a relaxed abdominal wall, emaciation (Rumpf) and descent of the diaphragm favor cardiac shift in the right and left lateral positions ("wandering heart"), elevation of the diaphragm, good nutrition, firm connective tissue and great vessels as well as increased pulmonary resistance (pulmonary passive congestion) oppose cardiac mobility. By extensive roentgenologic studies Zdansky confirmed the clinical findings of Determann whose results were also substantiated by Goldscheider

The highest degree of lateral cardiac mobility is found in asthenic individuals with constitutionally lax muscles and a flaccid vascular and connective tissue apparatus, in emaciated subjects disappearance of fat also contributes to poor support of the abdominal and chest organs, after several pregnancies or shortly post partum, weak abdominal walls permit marked displacement of the abdominal viscera and of the diaphragm and, thereby, lessen cardiac support, finally, considerable lateral mobility is also encountered when tonus of skeletal musculature is reduced

The lateral shift which amounts to 1 5 to 3 0 cm. according to clinical reports (Determann, Gerhardt, Hoffmann and Goldscheider) may reach 5 cm in these cases. Shifts of 8 to 9 cm , reported as maximal values on the basis of displacement of the apical impulse and of cardiac dullness, were never observed fluoroscopically by Zdansky.

Abnormally slight or even absent lateral mobility of the cardiac shadow is encountered with elevation of the diaphragm, large hearts, acute and chronic pulmonary congestion from heart disease, extreme emphysema, extensive pleural adhesions, hydrothorax, hydropericardium, and pericardial adhesions (Zdansky).

7. The Anterior View of the Heart in the Right and Left Lateral Recumbent Positions

The cardiac shadow experiences very striking alterations in the right and left lateral recumbent positions These changes involving position as well as shape are essentially effects of gravity Owing to gravity the mediastinum sinks to the side on which the patient reclines Consequently the diaphragm on this side ascends into

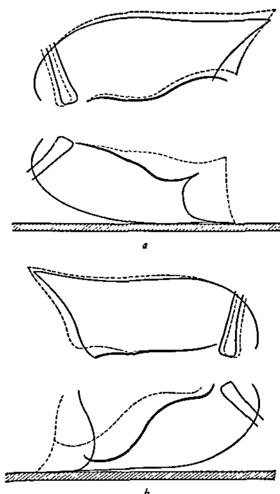


Fig 31 —Respiratory wandering of mediastinum and alterations of cardiac shape from respiration. (*a*) Right lateral position, (*b*) left lateral position. (______ average breathing, ------ deep inspiration.)

the thoracic cavity while the dome on the upper side descends and flattens (fig. 31a and b).

The descent and flattening of the upper diaphragmatic dome is produced by the reduction of intra-abdominal pressure on this side, conditioned by gravity, and by the pull exerted by the heart sinking into the lower side; the elevation of the lower dome of the diaphragm is explained by the augmented intra-abdominal pressure on this half and to relaxation resulting from descent of the heart.

II. Pulsations of the Cardiovascular Shadow

Observation of the cardiovascular pulsations is an important feature of every roentgenologic examination of the heart.

With the unaided eye only coarse movements are perceived. For their finer analysis, films in various phases or graphic registration of their course is necessary. Eijkman, Weber, v. Elischer, Th. and F. Groedel, Huismans, Cottenot, McPhedran and Weyl, Hirsch and Schwarzschild, Ludwig, and Eggli, among others, rendered a great service in devising apparatus which made it possible to obtain records at optional phases of cardiac activity or, at least, to permit the subsequent arrangement of the views obtained in accordance with definite phases. Then, it became feasible to undertake an exact analysis of the alterations of cardiovascular size and shape during pulsations. More recently roentgen cinematography, introduced by Kastle, Rieder, and Rosenthal has developed further in the hands of Chamberlain and Dock, Janker, Reynolds, and others, to open a vast field. Particularly by indirect roentgen cinematography, moving pictures of fluoroscopic images have made it possible to explain the anatomic features of some pathologic hearts and complicated congenital anomalies. Finally curvilinear registration of movements by roentgen kymography (p. 9) has made some contribution to the analysis of cardiovascular movements

1. Pulsations of the Left Ventricular Arc

If we consider at first the pulsations of the left ventricular arc on the fluoroscopic screen, we recognize that the medial movement, corresponding to the brevity of the systolic phase, evolves much faster than the diastolic lateral movement The pulsations are almost convergent toward the center of the cardiac shadow Usually, however, they are larger near the apex than at the base (Huismans, Schwarz, Dietlen, Laurell). This difference in the breadth of excursions is very striking in pendulous hearts in which the apex often executes systolic pulsations of astonishing size, directed toward the base (p. 103)

In many cases, however, the pulsations in the upper part of the left ventricular arc are larger than those of the apex

On the basis of surface kymographic studies, Stumpf distinguishes pulsation type I and II, depending on whether the apex or parts near the base of the left ventricle show the largest excursion of the visible pulsations In general, type II is less common than type I but becomes more frequent with increasing age Even in young patients it should be found in 60 per cent if the series contains those with oblique hearts Moreover, it appears more commonly in hearts above average in size and with a slow rate (under 70 per minute)

Stumpf is inclined to connect these two types of visible pulsations in some cases with different anatomic states of single sections of the ventricular wall Thus, type II is alleged to occur when there is a "special strength of the muscles of the outflow tract and weakness of the apical section" Since, however, they are also observed in patients in whom there is no reason to suspect that the myocardium is pathologic in any respect, Stumpf assumes other reasons for its appearance Since hearts displaying type II pulsations may show type I during exertional tachycardia with expiratory elevation of the diaphragm in the left lateral position and since in youthful individuals type II can be replaced by type I after exertion, Stumpf assumes that a variable position of the heart, the function of different groups of muscles, and the anatomic structure of the heart may determine the

With deep respiration one can observe the movements of the diaphragm and mediastinum. These are essentially different from those noted in the erect position (fig. 31a and b). The hemidiaphragm of the upper side, already flattened in quiet respiration, becomes more tense with deep inspiration so that it becomes even flatter without, however, much additional descent, sometimes it even rises as the lateral chest wall is lifted in inspiration The elevated hemidiaphragm on the lower side executes, *on the contrary*, a very large movement caudad. Coincident with this displacement of both diaphragmatic domes, the mediastinal shadow swings so that both of its borders shift toward the upper side. This respiratory mediastinal wandering (Holzknecht and Hofbauer) shows a smaller excursion in the region of the vascular band than in the heart for the following reason inspiratory contraction of the upper hemidiaphragm draws the central tendon toward the upper side, the contraction of the powerful lumbar sections of both hemidiaphragms draws it caudad and toward the midline. Consequently the heart also moves toward the midposition, at the height of deep inspiration it attains approximately the position held in the erect posture

The impression of wandering is heightened by the fact that the markedly rounded lower border of the heart deformed from the damming of blood, stretches and flattens in inspiration, consequently it withdraws farther from the lower chest wall than corresponds to the extent of the total cardiac shift within the thorax. Accordingly, inspiratory shift of the adjacent cardiac border should not be considered entirely an expression of mediastinal wandering but partly the result of flattening and downward pull In examining for respiratory mediastinal shift in the two lateral positions, less attention should be paid to the movement of the lower border of the heart than of the upper

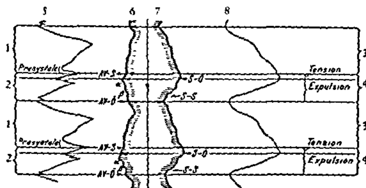
The greatest inspiratory shift is usually executed by the left cardiac border in the left lateral position since it represents the most mobile part. The smallest movements are executed by the right cardiac border in the left lateral position since the adjoining fixed caval axis allows relatively little change in position. With corresponding depth of respiration, the extent of mediastinal wandering in both lateral positions depends upon the degree of lateral cardiac mobility. A heart fixed within the thorax also loses respiratory mobility Therefore Zdansky recommended observing respiratory shift as a test of passive cardiac mobility (p 337) This requires none of the laborious linear measurements of positional alterations of the heart which are associated with various sources of error. Only the presence or absence of motion is utilized and is readily perceived since the eye is extremely sensitive to movement

In its wandering the cardiovascular complex changes shape (fig. 31a and b) and this may be considered a return, more or less complete, to its form in the erect position

The greater the lateral shift of the heart is and the more that spatial relations in the thorax are altered in lateral positions, the more extensively the cardiovascular complex changes, provided that conditions are otherwise the same The plasticity of the heart has great significance. a thin-walled heart, poorly filled with blood, changes its shape much more than one which is well filled, thick-walled, or even hypertrophied.

combination of systolic rotation of the entire heart, of pulsatory volume alterations of the left ventricle and left atrium and changes of cardiac form during a cycle

Kymographic analysis of the excursions of the left ventricular arc (Zdansky and Ellinger) has shown that these are not absolutely equal in all parts although they show extensive agreement with the Straub curves of ventricular volume over wide sections. At first one recognizes in the kymogram (fig. 7, 8, 32) the longer, flat increase of the diastolic outward movement Immediately before the systolic decline, a small plateau (VS-SÖ) usually corresponds to the presphygmic tension period, that is, to the beginning of systole Sometimes it shows a slight ascent which Westermark believes is due to raising of the heart (Ludwig) The shorter and more oblique descent of the curve corresponding to the ejection phase shows a constant notch,



of semilunar valves

1 Atrial emptying
2 Atrial filling
3 Ventricular diastole
4 Ventricular systole
5. Esophageal cardiogram (Edens)
6 Roentgenkymogram of right atrium (Zdansky and Ellinger)
7 Roentgenkymogram of left ventricle (Zdansky and Ellinger)
8 Ventricular volume curve (Straub)

an expression of the so-called initial oscillation, finally it ends in a small crest (SS) which marks a slight increase of ventricular volume from the closure of the semilunar valves. The brief valley in the curve corresponds to the isometric period when all valves are closed The diastolic rise shows small irregularities and moreover, a steeper part which indicates sudden enlargement of the ventricle from the influx of atrial blood as well as a flattening or indentation which may be, perhaps, an intrinsic oscillation of the ventricular wall resulting from the inrush of blood, there is a final oblique rise to the diastolic crest Almost all good long kymograms reveal these details which, by agreement with Straub's curves of ventricular volume (fig. 32), show that they represent primarily a curve of ventricular filling Kymographic investigations by Cignolini, Delherm and collaborators, Stumpf, by Cottenot and Heim de Balsac, and Westermark as well as the electrokymographic findings of Luisada and Fleischner, of Deutsch and coworkers have confirmed, with minor deviations, the reports of Zdansky and Ellinger.

appearance of both types of pulsation Type II is also found often with left heart hypertrophy and with athletic hearts, perfectly capable of function, and indeed with greater frequency when the heart is enlarged Reindell showed that *when these hearts were subjected to exercise tests* (bending the knees, running) type II tended to be transformed into type I He concluded that diminutive pulsations in the parts near the apex were conditioned by the following situation · at rest the sections near the base participated chiefly in systolic emptying while the region near the apex stowed away the augmented amount of residual blood Only with exercise was the residual blood expelled from the sections of the ventricles near the apex by more powerful contractions in this area, hence the increase of pulsations near the apex A persistence of type II in exercise would then imply a deficient capacity for contraction of the ventricle and permit, with plausibility, an inference of myocardial insufficiency On the other hand, Reindell like Stumpf ascribes no pathologic significance to a transient type II

The breadth of pulsations of the left ventricular arc is reported very differently and usually too high since ordinarily the statements involve fluoroscopic observations with short distance examination or measurements with a short target-film distance, that is, with central projection Stumpf determined kymographically the maximal, minimal, and average breadth of excursions of the left cardiac border for different ages, weights, heights and chest circumferences The values found were remarkably high although the curves were obtained with a long target-film distance In orthodiagraphy one is surprised at how small the excursions actually are when the pulsations are very striking on the screen. In the middle of the left ventricular arc, with an average rate and moderate depth of respiration, they do not exceed 2 to 3 mm. With bradycardia (Hoffmann, Dietlen), in some valvular lesions and congenital anomalies, in thyrotoxicosis (Moritz), with reduced inflow into the heart (Laurell), with pneumothorax and pneumopericardium they can be much larger, with left ventricular failure and higher grades of tachycardia they may be smaller The breadth of visible excursions often depends strongly on respiration (Dietlen) *and this is particularly distinct with marked respiratory arrhythmia.* The prolonged diastolic filling period of the heart during expiratory bradycardia increases the stroke volume and consequently the breadth of left ventricular excursions while the shortening associated with inspiratory acceleration has the opposite effect Moreover, inspiratory tension of the pericardium may contribute to the reduction of excursions. Sometimes, however, in inspiration the pulsations enlarge (Stumpf, *Westermark*).

In fluoroscopic observations one often gains the impression that the inward systolic movement of the left ventricular arc is not simultaneous in all parts Some authors believe that this movement proceeds as a sort of contraction wave from the cardiac base to the apex (Schwarz, von Criegern, Dietlen, Bayliss and Starling), others doubt the existence of such a course (Vaquez and Bordet, Laurell), still others saw, on the contrary, systolic inward movements which started earlier at the apex than at the base of the left ventricle (Gott). On the basis of precise electrokymographic registration it was actually demonstrated that in some cases a contraction wave seemed to proceed toward the cardiac base (Engstrom, Kjellberg, Persson and

rather it is determined by the individually different and remarkably complicated

combination of systolic rotation of the entire heart, of pulsatory volume alterations of the left ventricle and left atrium and changes of cardiac form during a cycle

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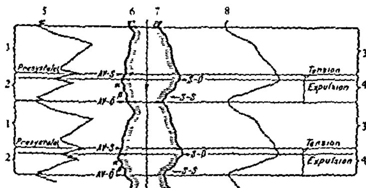


FIG 32.—Contrast of typical ventricular and atrial kymograms on one hand and the ventricular volume curve and esophageal cardiogram on the other AV-S = closure of atrioventricular valves, AV-Ö = opening of atrioventricular valves, S-S = closure of semilunar valves, S-Ö = opening of semilunar valves.

1. Atrial emptying
2. Atrial filling
3. Ventricular diastole
4. Ventricular systole
5. Esophageal cardiogram (Edens)
6. Roentgenkymogram of right atrium (Zdansky and Ellinger)
7. Roentgenkymogram of left ventricle (Zdansky and Ellinger)
8. Ventricular volume curve (Straub)

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Excursions of the left ventricular arc naturally are not determined exclusively by fluctuations of left ventricular volume. Undoubtedly they are also influenced by alteration of right ventricular volume, by rotation and transformation of the heart during its activity and they are modified in various uncontrollable ways. Finally, all those limitations which are valid when conclusions obtained from roentgenkymographic size of excursions are applied to the true size of excursions and movements of superficial points of the heart (p 9ff), should be recalled, then it should be clear that the kymogram can show only approximately parallel fluctuations of ventricular volume. It should also be clear that the kymogram at various places along the left ventricular arc shows different depths of excursions and also qualitative differences, thus, the systolic trough or the diastolic crest is sharper or flatter and details are evident in one place while they are missed in others Actually, under normal conditions, a reversal of the direction of the excursion can occasionally be demonstrated in parts of the ventricular arc near the base an outward movement during systole and an inward movement during diastole Zdansky and Ellinger interpreted these incidental systolic lateral movements as an expression of systolic cardiac bulge or lifting of the apex by Ludwig's lever movement. Fingerhuth and Bickenbach occasionally observed a reversal of ventricular pulsations which they ascribed to positional changes of the heart from respiration Reindell interpreted this reversal of apical pulsations as a result of a pendulum movement of the heart directed to the left predominating over the movement of systolic contraction along the left cardiac border. This does not, however, fully explain the phenomenon in all cases, therefore Reindell had recourse to additional hypotheses, the validity of which require further confirmation He found such lateral systolic movements frequently in the lower left cardiac border in trained athletes. In these individuals these pulsations vanished with exertion and gave way to normal systolic inward movements. On the other hand, when such pulsations are produced by myomalacia at the cardiac apex, they should be more distinct in an exercise test

These observations indicate that it is improper on the basis of the presence of lateral systolic movements of parts of the left ventricular arc near the apex to conclude without further ado that this section is abnormally distensible or that local passive dilatation has been produced by a myocardial infarct (Stumpf) or the presence of a completely unestablished "apical atony" (Bohme)

The breadth of excursions in a ventricular kymogram consequently is not a reliable measure for the stroke volume of the left ventricle The stroke volume does not depend to a great extent upon the pulsations of the lateral ventricular wall but much more on the excursions of the atrioventricular septum (Schwarz, Sundberg, Laurell), the latter are not susceptible to direct roentgenologic observation. The following observation suggests how little reliance can be placed upon conclusions about stroke volume based upon the pulsations of the left ventricular arc: precisely [illegible]

[illegible]
the systolic shift of the atrioventricular septum toward the cardiac apex is hampered and therefore the lateral wall of the ventricle together with the cardiac apex must in turn move toward the atrioventricular septum. Moreover the tonus of the myo-

cardium, the height of intrathoracic pressure and the inelasticity of the pericardium may influence the breadth of excursions and the course of visible and recordable pulsations.

There are also factors which confer different characteristics on the visible pulsations so that "strong and weak" types (v. Criegern), "stroke and pump" movements (Schwarz) and "excited, powerful and weak action" type (Dietlen) are mentioned. To these peculiarities of pulsations which contain some subjective features and hence are not susceptible to complete separation we shall return in connection with the conditions and diseases in which they are encountered. They differ not only by varying breadth of the excursion but also in the course of their in- and outward movements.

Finally it is obvious that a large heart will show smaller pulsations than a smaller heart with the same stroke volume.

These remarks do not deny that, *ceteris paribus*, the breadth of the excursion of the left ventricular arc parallels the stroke volume of this ventricle So many other factors influence the size of the visible pulsations, however, that extreme reserve is in order in evaluating the pulsatory movements in terms of stroke volume

2. Pulsations of the Aortic Knob

The pulsations of the aortic knob are opposite those of the left ventricular arc (figs. 5, 6, 8 and 33) The former, as typical arterial pulsations, are recognized by a brisk systolic outward and a slow diastolic inward movement Their kymographic registration yields essentially a pressure curve of the aortic lumen (Stumpf, Fetzer, Schilling, Zdansky and Ellinger, Cignolini, and others) According to Stumpf and Fürst, the size of the excursion amounts to about 2 mm with an average level of the diaphragm and a 2 meter target-film distance. With increased stroke volume of the heart they definitely enlarge There is, however, no strict parallelism with the stroke volume of the heart or blood pressure amplitude because pulsations of the aortic knob are more or less magnified by the total shift upward, to the left and backward, which the aortic arch experiences with every systole (Vaquez and Bordet, Dietlen, Zdansky and Ellinger). This shift which has been recorded kymographically on the ascending aorta (Bickenbach) is a normal event, it should not be considered a sign of abnormal thickness or rigidity of the aorta as has been repeatedly reported A good idea of how much the aortic arch shifts is provided when the esophagus is filled with barium Then, the section of esophagus which crosses the aortic arch executes a movement to the left each time the aortic knob moves laterally in systole. Since the esophagus and aortic arch are in close apposition at the site of crossing one may assume that this systolic shift of the esophagus practically corresponds to the shift of the arch (Zdansky and Ellinger). Weltz has shown that this may vary greatly. Elevation of the diaphragm and elongation of the aorta may enlarge it while descent of the diaphragm tends to diminish aortic shift and consequently reduces demonstrable pulsations of the knob

No reliable conception of the actual size of the pulsatory variations of aortic diameter can be obtained from the width of excursions of the knob. Only simultaneous kymographic registration of the shift of the barium-filled esophagus shows

this. The difference between the breadth of excursions of the aortic knob and the aortic bed of the esophagus alone gives approximately the actual size of the pulsatory variations of aortic diameter (Zdansky and Ellinger).

With this procedure the pulsatory alterations of aortic width show, on one hand, no strict parallelism with the stroke volume, nor on the other, with blood pressure amplitude This is explained by the influence of the anatomic and functional *status* of the aortic wall Thickening and rigidity of the aorta as well as a hypertonic orientation of its muscular elements tends to reduce the width of aortic pulsations

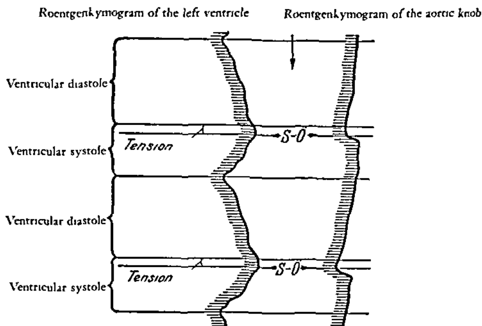


Fig. 33.—Contrast of ventricular and aortic kymograms S-O = opening of semilunar valves

while a hypotonic status tends to enlarge it (Zdansky). Weltz did not consider the dependence of this width on the tonus and muscular elements of the aorta in his criticism of our findings which, therefore, need not be regarded as implausible.

3. Pulsations of the Pulmonary Arc

Naturally the pulsations of the pulmonary arc are directed like those of the aortic knob although normally they are smaller Moreover, they certainly should not be considered a true expression of an alteration of width of the vessel since the pulmonary artery is shifted by the pulsation of the ventricle and the closely adjoining aorta.

4. Pulsations of the Left Atrium

Between the pulmonary arc and the arc of the left ventricle is a field, the anatomic "substrate" of which probably is not uniform, in some cases it is formed by the left atrium and in others by the pulmonary conus. Often this section seems im-

mobile to the unaided eye (Vaquez and Bordet). Possibly the atrium, distended systolically, appears in the space left open by the contracting ventricle (Bohme), so that the inward movement developing from ventricular contraction is balanced by the appearance of the enlarging atrium. Frequently, however, lateral pulsations are distinct, they alternate with those of the left ventricular arc and are smaller than those of the adjacent pulmonary arc Assmann and Dietlen believe a presystolic indentation occasionally is detectable. Whether this observation is reliable, however, remains dubious, for one will recall how slight a reduction of volume is produced by presystolic atrial contraction. Kymographic records of the excursions of the left atrial arc (Stumpf and Fetzer) revealed single elements of atrial activity only on some occasions, for example, sometimes there was an outward movement in the period of tension or an inward one at the beginning of ventricular diastole, otherwise the pulsations of the adjacent left ventricle or pulmonary artery often exerted a dominating influence on movements in this section Delherm was unable to record any characteristic pulsations. Kottgen believed an inward movement of the upper part could be seen during atrial systole Recently Fabicius found that median as well as lateral excursions might appear at the moment of ventricular contraction, he concluded that the atrium executes no intrinsic contractions but merely served as a complementary space to ventricular contractions

5. Pulsations of the Right Cardiac Border

Pulsations of the right border of the heart have always aroused special interest With the unaided eye it was believed that in some cases a systolic inward movement could be observed over its entire extent, synchronous and in unison with movements of the left ventricular arc In other cases, again, the right cardiac border bends laterad with each systole so that the entire cardiac shadow seems to oscillate (Th and F Groedel) Finally, with each systole, outward lightning-like movements often follow each other in the cephalic part of the right border, they have been designated "double beat" and correctly considered an expression of atrial activity (Dietlen, Assmann) Likewise a presystolic twitch has been reported (Arnsperger, Groedel). In the caudal part, pulsations generally are quieter than in the cranial section and show more or less an outspoken ventricular character Recurrently this has been submitted as evidence that the section of right border near the diaphragm is formed by the right ventricle Assmann and Laurell stress, however, that this conclusion is not compulsory since the section of atrium adjacent to the atrioventricular septum is affected by ventricular pulsations and accordingly may exhibit features of ventricular contraction Therefore one must anticipate a very gradual transition of atrial pulsations into ventricular ones

Kymographic registration of the pulsations (Zdansky and Ellinger) confirmed this In a vast majority of cases the pulsations of the cephalic parts of the right border did not represent pure volume curves of the atrium for they were influenced to a variable extent by the ventricular pulsations A very constant feature of the curves (figs 7 and 32) is a more or less sharp rise in the presphygmic period of tension and a steep fall at the beginning of the diastolic outward movement of the left ventricular arc. The rise corresponds to the beginning of atrial filling, the fall to the emptying

of the atrium in ventricular diastole. It is noteworthy, however, that this fall is not maintained throughout diastole, rather it is soon interrupted by a new rise. Undoubtedly this is a result of the ventricle widening in diastole and pressing the atrium laterad. Only in presystole the slope shows a small notch which is considered an expression of presystolic atrial contraction. In the systolic filling phase the slope is not uniform but rather shows two indentations (α and β) which probably result from suction on the atrioventricular septum bending it toward the apex during systole This suction (Rein) was observed in kymograms by Bohme who introduced opaque material directly into the two cavae. He observed an intermittent advance of blood containing the opaque agent toward the heart with each systole

This form of atrial curve explains the visual impression of the "double beat" on the right cardiac wall, as well as Groedel's pendulum movement of the heart The "double beat" is observed when the phase of systolic atrial filling finds strong expression in the pulsations of the right cardiac border. Actually this phase shows two peaks: the first is the sudden beginning of filling during the period of tension, the second, the maximum of right atrial filling immediately before the atrioventricular valves open, is designated AV-Ö. The impression of a pendulum movement of the heart is created when the phase of sudden diastolic emptying of the right atrium appears distinctly on the right cardiac border.

The closer one approaches to the cardiodiaphragmatic angle, the more ventricular pulsations tend to dominate the kymogram; finally no atrial pulsations are seen, or only some minor evidence of them remains, like the brief fall as the right atrium begins to empty Such residual traces of atrial pulsations are no longer discerned on the screen or in compressed curves of the surface kymogram but they may be preserved in long strip, detail-rich tracings.

When the entire right cardiac border seems to perform ventricular pulsations, precise kymographic registration usually reveals elements of atrial pulsations in the cephalic part.

Actually atrial and ventricular pulsations gradually merge. No sharp differentiation can be made Reports concerning the frequency and extent of participation of the right ventricle in the formation of the right cardiac border (Arendt and Baumann, Wilke, Fetzer) have not given these facts sufficient consideration.

6. Pulsations of the Right Side of the Vascular Band

The pulsations of the right border of the vascular band are usually complex. Often the typical undulating pulsations of the superior vena cava are seen even on the screen. Kymographic registration yields curves closely resembling Wencke-[illegible] and in place of the carotid wave they show a corresponding aortic [illegible] led [illegible] og-raphy reveals that these lateral movements tend to occur earlier than those of the aortic knob, the left ventricle, becoming larger as it fills, rises with a jerk at the moment of tension and lifts the ascending aorta to the right (Weltz); consequently the inward diastolic movement of the right vascular border is prematurely blocked and it even moves outward before systole.

Even pure arterial pulsations are not conclusive evidence that the aorta actually forms the right vascular border, the venous pulsations can be completely superimposed by pulsations from the aorta

7. Pulsations of Pulmonary Structures Adjoining the Heart

The hilar and lung shadows adjacent to the heart and great vessels show transmitted pulsations. They move in the same direction as the pulsations of the adjoining mediastinal shadow but become progressively smaller as the distance from the heart and great vessels increases Most striking are the pulsations of the left hilus which is correspondingly displaced with each systolic lateral movement of the pulmonary arc and the aortic knob (fig 5)

These transmitted pulsations differ from systolic intrinsic pulsations of the hilar shadows and of the large perihilar vessels which normally can be detected only by roentgenkymography or by a comparison of films made in systole and diastole (Cottenot). On the screen they are distinct only when abnormally increased, this happens, for example, when the pressure amplitude in the pulmonary artery increases as the result of a patent ductus Botalli, a septal defect, pulmonary regurgitation, thyrotoxicosis, or right ventricular hypertrophy (mitral valve disease, emphysema). Intrinsic pulsations are characterized by systolic increase and darkening of the hilar shadows, elongation and expansion of the vascular strands, and systolic enlargement of the vessel's cross section in the immediate vicinity of the hiluses

8. Pulsations of the Cardiovascular Shadow in Oblique Positions

In the left anterior oblique position oppositely and upwardly directed systolic pulsations are seen in the region of the two ventricles, that is, in the diaphragmatic parts of both cardiac borders. In the upper left cardiac contour these pulsations are gradually transformed into a rolling systolic pulsation directed dorsad In kymograms, Zdansky and Ellinger recognized them as atrial pulsations showing thorough agreement with esophageal cardiograms In contrast to the kymogram of the right atrium, the left atrial curve has a sharper and higher rise which might be connected with the flow of blood into the smaller left atrium (Zdansky and Ellinger), this is distended more briskly and strongly than the larger right atrium (Straub) Likewise in the cephalic section of the right cardiac border, atrial pulsations often are perceptible. No sharp distinction of ventricular from atrial pulsations is possible, however, on either the right or left border of the cardiac shadow, the pulsations merge into each other

In the right anterior oblique position, pulsations of the left atrium are easily recognized on the posterior cardiac wall These pulsations are, of course, influenced by the excursions of the atrioventricular septum like those of the right cardiac border (p. 59). The pulsations of the posterior border of the heart are rendered clearer when the esophagus is filled with barium, then pulsations of the posterior surface of the heart are transmitted to the adjacent esophagus This makes possible the recording of the esophageal cardiogram

In both oblique positions the ascending aorta pulsates distinctly and shifts in toto Bickenbach made roentgenkymograms of the aorta in the oblique positions in order

to determine the stroke volume with Brömser's method; naturally this procedure seems questionable considering that unequivocal demarcation of the aorta is frequently difficult in oblique positions.

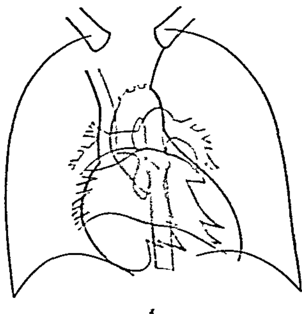
III. The Dynamics of the Heart in the Roentgen Image

Essentially more precise information on the inner dynamics of the heart than is provided by fluoroscopic observation or kymographic registration of the cardiovascular pulsations is yielded by serial- (Lind and Wegelius) and cineangiocardiography (Janker). They afford incomparable insight into the course and extent of the movements which take place within the heart during a cardiac cycle Since they reveal the changes in size, form, and reciprocal relations of single sections of the heart, they disclose the mechanism and dynamics of the pump and suction action of the heart better than any other method of investigation (Böhme).

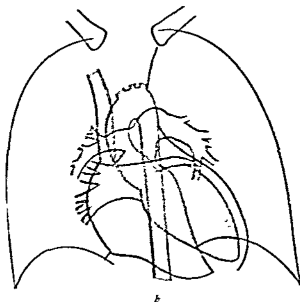
In order to show the reciprocal relations and transformations of the right and left hearts during the cardiac cycle, Zdansky superimposed the dextro- and levograms obtained with posteroanterior projection each time in the proper phases At the height of systole (fig 34a) both ventricles show maximal narrowing and in the apical portion they are scalloped and wavy from the protrusion of the papillary muscles. In this projection the right ventricle seems definitely smaller than the left and certainly smaller than it actually is because its lumen is reduced in systole to a small shell-shaped cleft which embraces the left ventricle from the right in front, and consequently it is penetrated almost on end with dorsoventral projection. The atria are maximally dilated and one perceives their great capacity Between the right atrium and the narrowly contracted, steeply rising outflow tract of the right ventricle a deep constriction is seen, corresponding to the crista supraventricularis This massive saddle-shaped bridge of tissue at the base of the right ventricle cuts in deeply in systole and contributes very definitely to narrowing of the right ventricle whereby the closely contracted conus pulmonalis shifts to the left

At the height of diastole (fig 34b) the engorged ventricles appear smooth and regular since the papillary muscles are now surrounded on all sides by blood containing opaque material. The auricular appendages are reduced by contraction, the right one is, on the whole, not defineable while the left lies like a small pennant on the base of the left ventricle The deep notch of the crista supraventricularis which was seen between the right atrium and the conus pulmonalis in systole, has completely vanished in the diastole of this particular case and the outflow tract of the right ventricle, becoming wide in diastole, now bends far to the right nearly to the superior vena cava This remarkable wandering of the conus and ostium pulmonale to the left during systole and to the right during diastole, which is augmented by systolic rotation of the whole heart (Janker), naturally also carries the trunk of the pulmonary artery with it with the following result. the reciprocal relation of the pulmonary artery to the aortic root is altered in such a way that the torsion of the great vessels around each other increases in systole and decreases in diastole.

Moreover the lateral excursions of the conus pulmonalis proceed opposite to the apical section of the right ventricle, that is, during systole in the apical part of



a



b

FIG. 34.—Projections of the dextrogram (blue) and levogram (red) in the anterior view. (*a*) At the height of systole. (*b*) At the height of diastole. (Reprinted from Zdansky, E. Zur Röntgenologie der Dynamik des Herzens. Fortschritte auf dem Gebiet der Röntgenstrahlen vereinigt mit Röntgenpraxis 75 1?0. 1951.)

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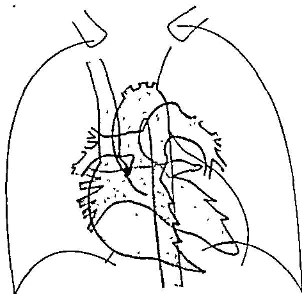
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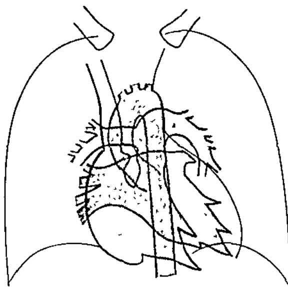
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Moreover the lateral excursions of the conus pulmonalis proceed opposite to the apical section of the right ventricle; that is, during systole in the apical part of



a



b

FIG 35—Projections of the dextrogram (blue) and levogram (red) in the anterior view. (*a*) At the beginning of diastole (*b*) At the beginning of expulsion (Reprinted from Zdansky, E Zur Rontgenologie der Dynamik des Herzens Fortschritte auf dem Gebiet der Rontgenstrahlen vereinigt mit Rontgenpraxis 75 180, 1951)

the heart the left ventricle increasingly appears on the anterior cardiac wall from the left-behind (Koch) and the right ventricle with its conus approaches the left cardiac border at the cardiac base.

Apart from this wandering in a horizontal plane, the conus and ostium pulmonale also show excursions in the craniocaudal direction since they are drawn toward the cardiac apex by the systolic contraction of the ventricle, to bend craniad again in diastole It is clear that the displacement of the pulmonary trunk toward the apex in systole opposes the pulsations of the pulmonary arc directed craniad and laterad and caused by the *systolic expansion and stretching of the pulmonary artery*, this exemplifies how complex the border pulsations of the cardiovascular shadow demonstrated in the roentgenkymogram are and the caution necessary in their *quantitative and qualitative interpretation*

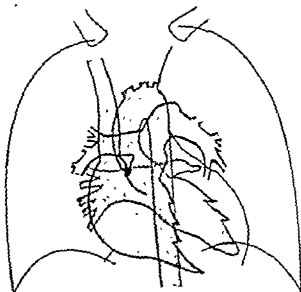
The excursions of the atrioventricular plane, so highly significant for the pump mechanism of the heart are naturally observed best on the right heart in the sagittal *cineangiocardiogram. One sees distinctly that the right atrium is actually distended* (fig 34a) as soon as the valve plane bends systolically toward the cardiac apex, this represents a very impressive picture of the suction action exerted on the caval blood *by the wandering of the valve plane* The heart is not only a pressure pump but a suction pump as well (Rein, Bohme)

Cardiac diastole begins very suddenly (fig 35a) and one sees with what remarkable speed the ventricles fill with rapid reduction of the atria and the auricular appendages although the scalloping of the projecting papillary muscles long remains visible. From the shortening and changing shape of the auricular appendages one gains the impression of their active contraction. The deep notch of the crista supraventricularis is distinctly more shallow and smaller The conus pulmonalis, already widening distinctly, begins to bend to the right. At the lower boundary of the left ventricle which belongs to the inflow tract of this ventricle, one perceives a bulge which is undoubtedly produced by the brisk surge of blood from the atrium into the relaxed ventricle in the first phase of diastole ("rapid filling").

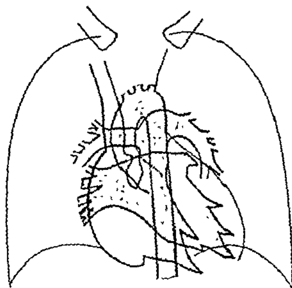
Soon after the beginning of systolic contraction of the ventricles (fig 35b) the scalloping of the papillary muscles appears. The conus pulmonalis begins to wander to the left and separates from the superior vena cava and the right atrium The auricular appendages fill The left shows definite differences of its shape in contrast to that in ventricular diastole. No outpouching of the inflow tract of the left ventricle like that seen at the beginning of diastole is found at any time during the ejection period. The pulmonary artery, especially the left, stretches in *systole* so that the vertex of the left branch appears higher. At the height of systole, however, the pulmonary artery is drawn in by systolic shortening of the ventricle and this opposes the rise of the pulmonary arc.

Thus analysis of the angiocardiogram illustrates the dynamics of the heart in an excellent way. Furthermore, it shows that no phase of systole exactly corresponds to any phase of *diastole in respect to the form of the individual cardiac chamber* although the filling of atria and ventricles may correspond approximately to each other. Rather, the ventricles and atria show a different shape even with the same content *according to the phase of ejection or filling* in which they are found.

In some respects mechanical or electrokymographic registration of cardiac pulsa-



a



b

FIG. 35 —Projections of the dextrogram (blue) and levogram (red) in the anterior view. (*a*) At the beginning of diastole. (*b*) At the beginning of expulsion (Reprinted from Zdansky, E Zur Rontgenologie der Dynamik des Herzens Fortschritte auf dem Gebiet der Rontgenstrahlen vereinigt mit Rontgenpraxis 75 180, 1951)

tions offers more than cineangiocardiography because they permit curvilinear registration of certain fine movements and their chronologic relations appear more distinctly and are more susceptible to more precise measurement. Thus, presphygmic tension time and the close of the semilunar valves are perceived in the ventricular kymogram but not in the cineangiocardiogram (p. 55). Moreover, the fact that the contraction of the right ventricle begins up to 0.03 second earlier than that of left ventricle was demonstrated only electrokymographically (Luisada and Fleischner).

IV. The Size and Shape of the Heart in the Newborn or Very Young Child

The heart of newborn or very young children (fig 36) shows marked individual differences and some special features when contrasted with the picture of later childhood and adults Usually the silhouette of the infantile heart shows less differ-

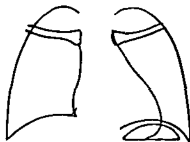


FIG 36 —Child's heart Girl, 5 years old (copy of film).

entiation, the supracardiac section of the mediastinal shadow is relatively broad, *short, and often lacks contrast to the cardiac shadow, the aortic knob* is flat or missing, the cardiac waist may be absent or merely suggested creating the impression of a mitral configuration These features are determined in part by the relative size of the right heart and the relative width of the pulmonary conus in the early months and years of life, in part by the special spatial conditions in the infantile thorax and by the size of the thymus

Kreuzfuchs traces the poor development of the aortic knob essentially to the absence of a sagittal section of the aortic arch in *the first few years of life, the course* of the arch through the chest is more oblique than later. He also accounts for the different course of the aorta by the right-sided position of the infantile trachea which allows the arch to pursue that course. Only as the trachea subsequently moves to the left and approaches the midline is the aorta forced to describe an angular kink from its almost frontal pretracheal course into a sagittal orientation in its distal section

[illegible] and but its shortness and rela-

into a transverse position, from the front the heart is oval and its angle of inclination is definitely less than 45 degrees. Moreover, the relatively great depth of the thorax diminishes the inclination of the heart from above and behind to below and forward,

the angle at which the diagonal diameter of the lateral view (p. 75) joins the horizontal is smaller than in later years. Moreover, the course of the large vessels is definitely influenced by the spatial conditions. The great vessels rise less sharply from the heart and move dorsad more abruptly. Therefore, in anterior views the cardiovascular complex is seen in projective shortening from below, the vascular band appears squatty and the pulmonary conus fills the cardiac waist more or less *completely*.

The thymus is very important in regard to the special form of the cardiovascular shadow in the early months of life. The gland may sit like a cap over the base of the heart and atria to simulate decided cardiac enlargement or a pericardial effusion, or *it may surround the great vessels or trachea in a cylindrical or bulbous form* (Gravinghoff, Hotz). In this way the supracardiac section of the mediastinal shadow may be widened, rise like a column over the heart, or extend as an arch or with

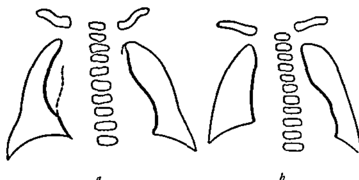


FIG 37.—Cardiovascular shadows in small children with thymic hyperplasia (after Hotz) (*a*) Nodular type of thymus in a boy 5 weeks old (*b*) Columnar type of thymus in boy 3 months old.

knobs into the lung fields (fig. 37a and b). Correspondingly, in lateral views a shadow is often visible in the upper section of the retrosternal space These pictures remain in the domain of the normal. With the combination of an enlarged thymus and a cervical goiter, cardiac enlargement appears (Hotz) and tends to be definitely pathologic

On the second day of life the cardiovascular shadow becomes smaller (Bell, Weymuller and Krahulick, Dietlen and Schall), this has been ascribed to a recession of the physiologic plethora of the newborn or a diminution of the thymus which swells at birth.

Beginning with the end of the second year the cardiovascular shadow progressively changes its shape, Hecht and Gravinghoff believe this ceases with the sixth year This change does not evolve with exact parallelism to the transformation of the thorax (Gravinghoff) but starts earlier Consequently one may infer that thymic regression plays a major role in this connection. With its diminution the aortic knob appears with increasing regularity on the left, on the right the transition of the laterally concave V brachiocephalica (anonyma) dextra into the superior vena cava appears with increasing regularity, consequently the vascular band gradually as-

sumes the typical adult form. In severe gastroenteritis of the small child the size of the heart may diminish considerably within a few days as the result of great loss of fluid (p. 131) and may approach the adult shape of the cardiac shadow. Particularly striking is the narrowing of the vascular band and the appearance of the cardiac waist. Sometimes, however, an adult type of vascular band is present even in newborn, indeed this may happen despite an abnormally enlarged thymus when the latter develops most in a posterior and longitudinal direction (Gravinghoff)

Besides regression of the thymus, transformation of the thorax plays a large part in the change of the cardiovascular shadow. With growth in length, the thorax progressively flattens since its sternovertebral diameter increases less than its transverse The transformation of the conical short thorax of a young child into the longer, flatter thorax of the adult causes the heart to descend from a transverse to an oblique position and reduces its inclination from above-posteriorly to below-anteriorly. With this alteration of position the cardiac waist becomes distinct for the first time, the aortic knob now protrudes and the indentations appear at the cardiodiaphragmatic angles. In the school age are found all cardiac shapes encountered in adults Even somewhat later, median placed hearts, with more or less decided orthostatic diminution of volume are very common. In obese individuals, transverse hearts are also seen Particularly in the female, the infantile heart shape rather often persists into the adult period, a situation which often may be ascribed, with some degree of probability, to the infantile, relatively short and deep thorax of these individuals (p 98)

The relations of the size of the childhood heart are less constant to the different body measurements than those of adults since cardiac development does not always proceed apace with the development of other bodily measurements (p. 78, 80)

V. Roentgenologic Measurements of the Heart

Different measurements of the orthodiagram have been reported to represent cardiac size numerically Many of these measurements were introduced by Moritz and are still used Measurements are suitable for numerically defining the size and to a certain extent the shape of the cardiac shadow more or less accurately.

The measurements of the cardiac shadow (fig 38) are

1. The mid-right and mid-left diameters (Mr and Ml). These are the greatest horizontal distances of the right and left cardiac borders from the midline The mid-*left diameter is lower than the right and sometimes it runs* even below the dome of the diaphragm since the left cardiac border may proceed laterad even lower, never-[illegible] the level of the [illegible] ng as the mid-[illegible] women 1.2 4 (Dietlen), and for children 1.2 2 (Veith)

The ratio of Mr:Ml depends decidedly upon the height of the diaphragm Owing to the obliquity of the cardiac axis, the mid-left diameter changes more than the mid-right from alterations of the diaphragm, with elevation, the ratio of Mr:Ml shifts in the direction of 1: > 2 and with diaphragmatic descent 1: < 2 (fig. 39).

2. The diagonal diameter or diagonal of the heart (D) (Wenckebach) This line joins the two most laterad points of the cardiac shadow, thus it joins the lateral ends of Mr and Ml.

3. The transverse diameter (Tr_H) represents the sum of the mid-right and mid-left diameters (Mr + Ml). The transverse diameter has attracted considerable attention although it is subject to great errors. Moritz, as well as Dietlen, Hammer, Weiss, and others, has shown that Tr_H is a very unreliable measure of cardiac size

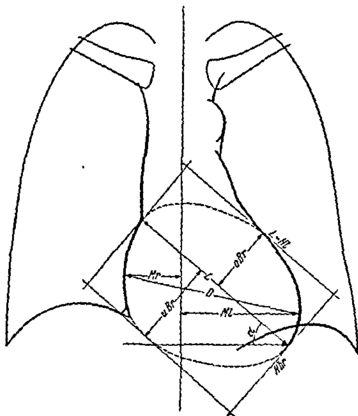


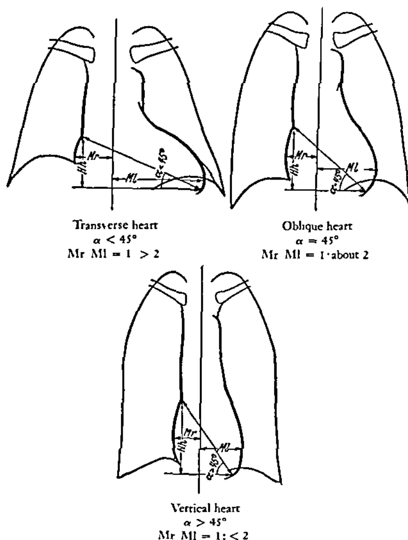
Fig 38.—Roentgenologic measurements of the heart Mr = mid-right distance, Ml = mid-left distance, D = diagonal diameter, L = longitudinal diameter (Hl), α = angle of cardiac inclination, oBr = upper cardiac breadth, uBr = lower cardiac breadth, HBr = cardiac breadth

since it depends to a great extent upon the angle of inclination (see below) (fig 39) The more vertical the heart (the greater its angle of inclination), the smaller Mr and Ml, and consequently the sum of these distances, becomes, as the heart becomes more oblique the angle of inclination decreases and these values increase (p. 71).

Despite this, Tr_H has excited great interest because it is easy to measure and agrees, in principle, with cardiac breadth as determined by percussion and with the anatomic transverse cardiac diameter. Moreover, in serial investigations average values change in the same direction as alterations in average heart size despite the sources of error (Kienbock, Deutsch and Kauf, Rautmann, Gotthardt, and others). In large series the errors are compensated since the deviations above and below are

cancelled For this reason only results based upon large series can be evaluated. This *also holds for the diagonal diameter (D) which agrees fairly well with* Tr_H although naturally it is somewhat larger.

Because of the various sources of error, Moritz always considered Tr_H merely an aid to preliminary orientation and recommended the following measurements for more precise investigation of cardiac size



Transverse heart
$\alpha < 45°$
Mr Ml = 1 > 2

Oblique heart
$\alpha = 45°$
Mr Ml = 1 : about 2

Vertical heart
$\alpha > 45°$
Mr Ml = 1 : < 2

FIG. 39.—Dependence of Tr_H (= Mr + Ml) and Hh on angle α of inclination of the heart.

4. Longitudinal diameter (L) or cardiac length (Hl). This is the distance from the right cardiovascular angle to the cardiac apex This measurement is sufficiently exact in most cases. As a rule, location of the right cardiovascular angle offers no problems but demarcation of the apex sometimes is difficult or impossible. The last situation occurs particularly in patients with very large hearts which sink deep in the abdominal shadow.

Measurement of the longitudinal diameter has an advantage over that of the transverse diameter in that it is affected less by the amount of cardiac inclination toward the horizontal plane. Since, however, the heart always experiences a definite projectional shortening toward the frontal plane with dorsoventral passage of the ray, depending upon the degree of its inclination, the longitudinal cardiac diameter need not equal cardiac length

Dietlen combined measurement of the longitudinal diameter with the transverse He considered these values most important and sufficient for practical purposes According to him they maintain a fairly constant mutual ratio Tr_B L = 1.0·1.1. Deviations from this ratio should indicate a pathologic situation

5. The angle of inclination (α) is the angle which the longitudinal diameter of the cardiac shadow (L) forms with the horizontal. Many consider it the most important characterization of cardiac position within the thorax According to its size, oblique, transverse, and vertical hearts are distinguished (fig 39) With an oblique heart the angle of inclination amounts to about 45 degrees, in the transverse heart it is smaller and in the vertical larger than 45 degrees Since the cardiac axis runs from the right-behind-above to the left-below-anterior, its obliquity in space is only imperfectly characterized by the angle of inclination, it is merely the inclination of the heart against the horizontal plane but not against the frontal plane; the latter can be recognized only with transverse passage of the ray

6. The broad diameter (Br) or cardiac breadth (Hbr) This is the sum of the greatest distances from the longitudinal diameter (L) to the right lower and left upper borders. The upper border (oBr) is easy to measure One drops a perpendicular from the notch between the left cardiac border and the pulmonary arc to L (Moritz), *this distance does not always correspond to the largest upper breadth* (Assmann) since marked rounding and bulging of the left ventricular arc may place the upper broad dimension nearer the apex

The lower width (uBr) is more difficult to measure since the turn of the right into the lower cardiac border is not always readily defined and the right cardiodiaphragmatic angle need not always correspond to the greatest distance of the lower border from L. Often, this is constructed in the region of invisible diaphragmatic border of the heart, as will be discussed later, by joining the right cardiodiaphragmatic angle and the cardiac apex by a flat, caudal-convex line. Since the lower width of the heart depends upon an imaginary line, cardiac breadth and all measurements involving this dimension are frequently rejected

The determination of cardiac breadth yields a second measurement which is fairly independent of the angle of inclination. Naturally this may also change somewhat when the heart rotates around its long axis as the diaphragmatic level changes and consequently presents a different breadth in the frontal plane These changes may, however, be only trifling. Moritz designated this measurement as "cardiac slenderness", it is the ratio of Hl to Hbr and is expressed by the quotient Hbr/Hl

7. The cardiac rectangle (Hr) is the product of the longitudinal diameter and cardiac width (Hl × Hbr) Consequently it represents an oblique rectangle corresponding to the angle of cardiac inclination and with its corners projecting beyond the cardiac shadow (fig. 40). Moritz regarded it as a bidimensional relative measure of cardiac size. The relation between the cardiac rectangle and cardiac surface,

measured planimetrically (see below) averages 1.31:1 in the erect and 1.33:1 in the horizontal position (Moritz), in other words the cardiac rectangle is, on the average, one third larger than the cardiac surface.

Although the size of the cardiac shadow is more perfectly defined by the longitudinal diameter and breadth than by simple measurement of Tr_{II} and Hl, the cardiac rectangle aroused only little interest. This depends partly upon the fact that its determination seems too complicated for routine use, moreover, some object that subjective judgments enter too much into the estimation (Otten, Kirsch, and others). The actual difficulties in measuring L and Br, indicated above, are always such that their general application seems inadvisable. On the other hand the experienced

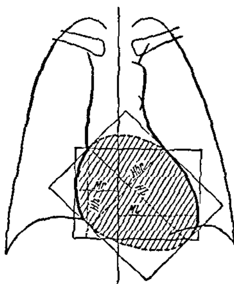


Fig 40.—Cardiac surface (hatched), cardiac rectangle (oblique rectangle), and cardiac surface rectangle (horizontal rectangle)

observer avoids crude errors and makes these measurements only when they can be done satisfactorily

Despite every precaution, evaluation of the *cardiac rectangle* as a *relative* measure of heart size actually contains all the sources of error mentioned earlier. They arise from the dependence of the longitudinal diameter on the inclination in the *frontal plane* and of the *cardiac breadth* upon *varying rotation* of the heart about its anatomic longitudinal axis. Furthermore, this measurement like many others employs a single projection to measure the size of an irregular three dimensional body.

8 Cardiac height (Hh). This is the distance from the right cardiovascular angle to a horizontal drawn through the cardiac apex (Kirsch) (fig 40). The measurement presents no difficulties if the angle and the apex can be defined well. As figure 39 shows, the angle of cardiac inclination influences cardiac height (Hh) in a sense opposite to the transverse diameter (Tr_{II}). As the angle of inclination increases, Tr_{II} becomes smaller and Hh larger. Kirsch introduced cardiac height

(Hh) to compensate for those errors in cardiac measurements arising from a dependence of Tr_H on the angle of inclination

9. Cardiac surface rectangle (Hflr). This is the product of the transverse diameter of the heart and cardiac height ($Tr_H \times Hh$) Kirsch recommends Hflr (fig. 40) as a relative measure of cardiac size since this rectangle is easier to construct and less subjective, moreover, its area agrees more closely with cardiac area than does the Moritz rectangle.

Actually the simplicity of determination is striking and the elimination of cardiac breadth from the calculation avoids the subjective factor attached to such determinations of surface. The average approximation of the cardiac surface rectangle to the surface measured planimetrically is for the moment open to doubt In a large series we found the average ratio of Hfl Hflr = 1 1 09 while the ratio of Hfl Hr was 1:1 37. The following point has great significance in the question of which of the rectangles is preferable as the relative measure of cardiac surface The average approximation to the absolute value of cardiac surface means less than the constancy of the ratio of cardiac area to size Obviously the ratio of the Moritz rectangle to the planimetric cardiac surface is definitely more constant than the Kirsch rectangle In a large series we calculated the extreme deviations of the ratio Hfl Hr = 1 max 1.39 to min 1 34 while we found in the ratio of Hfl Hflr a much greater deviation of 1·max 1 3 to min 0.93. Accordingly the Moritz rectangle is the preferable rectangle as a relative cardiac measure

10 Cardiac surface (Hfl) This is the planimetric surface of the sagittal orthodiagram (Moritz) * The determination of cardiac area requires some practice and yields satisfactory results in the hands of those expert in it Since cardiac shadows merge above with the vascular band and below with the abdominal shadow, their limits must be drawn (figs 38 and 40). Moritz connected the right cardiovascular angle with the upper end of the left cardiac border by a cranio-convex line, the place where right cardiac border bends into the lower border is joined to the cardiac apex through a flat caudo-convex line, separating the cardiac shadow above and below Owing to these arbitrary supplementations of the limits, measurement of cardiac surface has received some criticism Otten, Assmann, Kirsch, and others believe these supplementations are so conjectural and arbitrary that cardiac surface should be measured by a more precise method On the contrary Moritz, Dietlen, Geigel, Rohrer, Bernuth, Kahlstorf and Zdansky believe that knowledge of the anatomic relations plus a sense of proportion permits demarcation of the cardiac surface above and below and judgment of the actual situation with close approximation Such supplementation of the cardiac contours is justified—as Rohrer correctly stresses—because the caudal and cephalic borders of the heart are always obtained in approximately the same way, if the objection were sustained, one would be compelled to reject the value of measurements of the directly visible cardiac borders as relative measures of heart size We concur with Dietlen determination of cardiac size has value only when performed by a trained worker and that objections raised

* In the absence of a planimeter the cardiac area can be determined by the method of Geigel The orthodiagram is transferred to millimeter paper, cut out, and weighed When the weight of 100 cc of millimeter paper is known, the area of the cardiac surface cut out from the same paper can be calculated with sufficient exactitude

against its universal employment are valid because the correct drawing of the necessary supplements requires special training and experience. If these presumptions are fulfilled, cardiac area represents a very important cardiac measurement since it also opens *the possibility of determining cardiac volume by the Rohrer and* Kahlstorf methods (see below).

11. Depth measurement of the heart. All measures previously mentioned are based upon the sagittal orthodiagram. Determinations of size based upon these measurements alone presume that this projection has a fairly fixed relation to its anteroposterior depth This is, however, untrue even for the normal heart and for hearts uniformly enlarged, two hearts, equal in volume, may possess very different depths owing to their different positions in the chest (Kahlstorf, Roesler). Even with the same spatial conditions, the depth of equally voluminous hearts can differ and change *independently of the surface area of the sagittal orthodiagram* Undoubtedly the heart also suffers from changes in shape which are not conditioned by spatial intrathoracic relations (Zdansky). Consequently, it is impossible merely on the basis of the sagittal projection to form reliable conclusions on cardiac size. It is easy to understand that measurement of the sagittal projection of a pathologic heart is even less reliable than of a normal one (Assmann). One need merely recall the great increase of the depth diameter of the heart with mitral disease and a large left atrium

Accordingly, to determine cardiac volume, measurements of the heart's depth are indispensible. As early as 1904, Moritz studied this problem. In the frontal orthodiagram he used the greatest thickness of the heart, that is, the length of a line which generally starts where the cardiac shadow retreats from the anterior chest wall and ends at the most remote point on the posterior cardiac surface

For volume determinations Rohrer utilized the greatest horizontal a-p diameter *in the frontal orthodiagram* (lmax) *(fig. 41). This is the longest horizontal line* joining the anterior and posterior borders of the cardiac shadow. When it is difficult to define the posterior border of the heart, the place can be located indirectly by filling the esophagus with barium One should not distend the esophagus so that it indents the yielding posterior cardiac wall to make the depth diameter shorter than it actually is The coat of barium on the esophageal wall should just be visible (Zdansky).

H. Roesler rejected the Rohrer depth diameter. He stressed that with frontal passage of the ray, the most ventral point of the heart cannot usually be accurately located since the heart, bulging into the curvature of the anterior left chest wall, is projected into the sternum. Consequently the Rohrer depth diameter may be smaller than the true one. To avoid this error Roesler advocates a measure which is almost identical with that of Moritz.

Assmann described as a measure for the frontal orthodiagram, the diagonal diameter (D) and the absolute depth diameter (T) (fig. 41). The diagonal diameter designates nearly the axis of the lateral view and consequently runs from above-behind to forward-below. Its upper end lies at the junction of the following structures in front and above, the cross section of the pulmonary artery, below, the somewhat larger cross section of the upper pulmonary veins, and behind, the longitudinal division of the *descending bronchus which divides into numerous* branches running

in the path of the central ray precisely at this place on the hilus, these are recognized as clear, round spots. Its lower end lies in the angle between the diaphragm and the chest wall. Although this diagonal does not correspond to any anatomic cardiac measurement, still it may serve to obtain an idea of cardiac inclination toward the frontal plane (just as the longitudinal diameter of the sagittal orthodiagram shows the degree of inclination toward the sagittal plane). For the oblique heart it amounts to 45 to 50 degrees (Assmann).

Assmann measured depth extension of the heart by drawing two perpendiculars from the points on the anterior and posterior walls farthest from the diagonal diameter The sum of these perpendiculars, t_1 and t_2, provides the "absolute depth

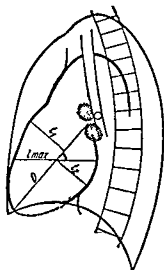


Fig. 41 —Depth dimensions of heart D = diagonal diameter of the frontal orthodiagram according to Assmann. $t_1 + t_2$ = absolute depth diameter according to Assmann lmax = greatest horizontal depth diameter according to Rohrer

diameter" The advantage of this measurement (Assmann) over the greatest a-p diameter (Rohrer) is its independence from the angle of cardiac inclination toward the frontal plane, Rohrer's depth diameter is a function of this angle of inclination. However, it is precisely this dependence upon the degree of cardiac inclination that makes Rohrer's *depth dimension acceptable for volume determinations of the heart* (see below).

12. The horizontal orthodiagram represents the greatest horizontal cross-section of the heart (Klason). It is constructed according to the model of Palmieri's radioplastic method whereby the patient is rotated on a swivel chair to obtain gradually the longitudinal axes falling through the heart around 360 degrees After each rotation through a few degrees the lateral contours of the cardiac shadow are recorded on roentgen paper from which the picture of the horizontal cross-section is gradually obtained.

13 Calculations of cardiac volume The volume of the heart can be determined roentgenologically in two ways by a reconstruction of a plastic model and deter-

mination of its water-displacement or by calculation from measurements of various projections of the heart. The methods of plastic reconstruction were discussed on page 7 They require special apparatus and demand so much time and effort that they are poorly adapted for serial investigations. It is much simpler to calculate cardiac volume from measurements of various cardiac projections. The different methods of calculation are based upon the principle that besides measurements on a parallel projection (as a rule, the sagittal orthodiagram), a depth diameter is included as the third dimension. One must realize that, at best, this provides only an approximation since the heart has irregular outlines with only a partial similarity to geometric structures for which these calculations are mathematically valid Nevertheless, long experience has taught us not to share Frik's pessimism, namely, that it is impossible to calculate cardiac volume with sufficient exactitude for practical purposes

Geigel's determination of cardiac volume was simply the first crude approximation and was considered as such by him By assuming that the heart is approximately spherical, its volume was calculated from Moritz's cardiac surface according to the formula $\text{Vol} = F^{3/2} \times \frac{4}{3\sqrt{\pi}}$ This formula which serves better as a three dimensional approximation than as a precise measure of cardiac size was employed by Geigel as a relative measure of cardiac size by omitting the constant $\frac{4}{3\sqrt{\pi}}$ and was related to body weight For the moment we shall not go further into this measure but will return to it in discussing the relation of cardiac size to different dimensions of the body

The formula of Salotti $\text{Vol} = \frac{a+b}{2} \times (a+b) \times (c+d)$ in which a + b indicates cardiac breadth and c + d, the cardiac length, likewise yields only approximate values

The claim for greatest possible exactitude is fulfilled in many directions by Rohrer's calculation He proceeds from the proposition that the volume of a regular object of optional form equals the product from the size of selected parallel projections and the average linear extent of the direction perpendicular to the first plane of projection He arrives at a simple determination of cardiac volume, in which the margin of error amounts to 10 to 15 per cent in most unfavorable cases on the basis of experimental cardiac models. In the Rohrer formula ($Fa \times lmax \times 0\ 63 = J$), J is the cardiac volume sought, Fa the planimetric surface as measured by Moritz (p 73), lmax the greatest horizontal depth diameter (p 75), and 0 63 a constant. The product lmax × 0 63 replaces the average linear extent of the sagittal dimension for the latter has a constant relation to the greatest depth diameter for any position of a regular object. Rohrer obtained the factor 0 63 empirically, it lies approximately between the factor for a transverse paraboloid and an ellipsoid Subsequently Kahlstorf* arrived at the same formula in another way

The reciprocal dependence of cardiac area and the greatest horizontal depth diameter on the position of the heart in space makes it necessary in every volume determination to obtain the two values in precisely the same position of the body To maintain the body in the same position, one should employ the lead crosses

* Ludwig used the cardiac rectangle in place of the cardiac surface (Fa) in the Rohrer formula This requires a substitution of the constant 0 46 for 0 63 The Ludwig formula is $V = R \times I_{90h} \times 0\ 46$ V is the cardiac volume, R, the cardiac rectangle of Moritz, and I_{90h} the greatest horizontal cardiac diameter of the heart

described on page 6 With frontal passage of the ray, if the transverse bars of both crosses lie at exactly the same level and perpendicular to the plane of the screen, the inclination of the upper half of the body is precisely the same as with sagittal passage of the ray and rotation is exactly 90 degrees Only when these precautions are strictly followed is the degree of exactitude obtainable within the limitations of the Rohrer method secured.

Roentgenologic calculation of cardiac volume is technically impossible when the apex cannot be defined and determination of the depth diameter is uncertain owing to obesity, pulmonary stasis, a large heart, or when breast shadows are projected into the anterior mediastinum. Volume determinations should not be attempted in such cases. Even if this limits the applicability of the method, its field of employment is still vast because these difficulties are unusual *in young individuals* who are precisely the most suitable for investigations of the physiology of circulation, moreover, in respect to athletics and ability to perform military service, it is often an excellent yardstick for expressing an expert opinion

With proper posing of questions and careful selection of patients or experimental subjects, the Rohrer method, *in our experience*, yields excellent results When properly performed, its superiority over all other methods for determining cardiac volume roentgenologically, resides in the fact that it provides with close approximation, a reliable volumetric measure of cardiac size, its disadvantage is that it cannot be used in all cases

When cardiac volume cannot be determined by the Rohrer formula, Ludwig calculates it, with good approximation, from the transverse cardiac diameter (T) and the greatest horizontal depth diameter (T_{90}) His formula is $V = 60 \times (T + T_{90}) \times 700$. This formula, whose chief attraction is its simplicity, yields unduly small values when there is marked cardiac enlargement Consequently Ludwig recommends the addition of 15 per cent when $T + T_{90}$ is above 25 cm. and 30 per cent when it is over 30 cm

A second formula (Rohrer) reported for determining cardiac volume calculates it from the surface of the sagittal orthodiagram (Fs), the surface of the transverse orthodiagram (Ft), and cardiac height (h) $V = \frac{Fs \times Ft}{h} \times k_3$, k_3 is a constant, 0 75 The formula found little acceptance because the transverse surface is determined with great difficulty.

Cignolini as well as Benedetti and Bollini utilize as a measure of cardiac depth, the breadth of the cardiac shadow in the left anterior oblique position. The Cignolini formula is

$$Vol = \frac{4}{3} \times \frac{S}{4} \times \frac{DPr}{2}.$$

S is the cardiac surface and DPr the dimension just mentioned. Benedetti and Bollini calculate the volume according to the formula Vol = DLo × DLa × DApO in which DLo is cardiac length, DLa cardiac breadth in the anterior view and DApO the breadth of the cardiac shadow in the left anterior oblique position

VI. Correlative Determinations of Cardiac Size

The main objective of roentgenologic cardiac measurements is to answer the question of whether the heart is normal, *too large, or too small* for the subject

To answer these questions, extensive studies were undertaken to ascertain the relation of average cardiac size to various dimensions, characteristics, and functions of the body (Moritz, Dietlen, Francke, Claytor and Merill, Otten, Hammer, Groedel, v. Teubern, Haudek, Veith, Geigel, Kirsch, Rautmann, Hecht, v. Bernuth, Salotti, Fray, Hodges and Eyster, Benedetti and Bollini, Kahlstorf, Cignolini, Ludwig, and others) It should be stated in advance that the expectations associated with these studies were realized only in part, not only due to the difficulties of precise roentgenologic determinations of cardiac size but also to the impossibility of including all those factors of body and peripheral circulation which decisively influence cardiac size.

Anatomic studies have shown that the state of the muscles and, thereby, the weight and to a certain extent even the size of the cadaver heart, depends primarily upon the condition of skeletal musculature, its extent and type of activity. "So all animals, man included, that have stronger and more sturdy frame, with large brawny limbs some distance from the heart, have a more thick, powerful, and muscular heart, as is obvious and necessary. On the contrary, those whose structure is more slender and soft, have a more flaccid heart, less massive and weaker, with few or no fibers internally" (Harvey). This precept enunciated in 1628 implies that the mass of the heart depends upon body build and more particularly upon the state of the body musculature. On the basis of exact heart weights, Hirsch reached the same conclusion Moreover, Kulbs demonstrated that a dog working on a treadmill has a larger heart than its control At present we know that cardiac volume, roentgenologically determined, need in no way keep strict step with the development of skeletal musculature since—as v. Weizsacker correctly noted—various working conditions place different loads on the heart and skeletal muscles (p. 122).

Anatomic cardiac measurements are not in full agreement with the proportions disclosed by the roentgenogram. Strictly speaking, they are not comparable to each other Cardiac size and shape change essentially after death owing to rigor mortis which empties most of the content of the left ventricle and some of the right (Rothberger, Aschoff, de la Camp), particularly with a large aortic heart there is a rather amazing reduction in size in the cadaver as compared to the living heart. While cadaver heart volume is determined largely by the status of its muscle, the living heart size is, in addition, decidedly influenced by its blood content which is subject to great variations depending upon circulating blood volume, cardiac rate, contraction force, diastolic myocardial tension, and the resistance against which the heart works.

In correlative determinations of cardiac size, every effort should be made to exclude as far as possible, all intra- and extracardiac factors which introduce fluctuations of cardiac filling Naturally this goal is never attained. It can be approximated, however, by making all determinations under standard conditions and maintaining them as constant as possible For reasons discussed later, cardiac measurements should be made at the same time of day, preferably when fasting, with no exertion in excess of the individual's average in the preceding twenty-four hours (Zdansky). Owing to static lability of cardiac size (p 94) it would also be desirable to secure cardiac measurements in the horizontal posture (Moritz, Dietlen). These precautions exclude important factors which could lead to incalculable alterations of cardiac filling.

For correlative determinations of cardiac size, two methods, different in principle, are employed: (1) empiric tabellar arrangement of various cardiac measurements to all values of one or several body dimensions with a statement of upper and lower normal limits, (2) empiric calculations of ratios (average values as well as their upper and lower normal limits) between various cardiac measurements and one or another body dimension.

Thus, various measurements of the sagittal orthodiagram (Tr_H, Hl, Hbr, Hh, Hr, Hflr, Hfl) are related to various absolute and relative body measurements (height, weight, chest circumference, transverse diameter of the thorax, body rectangle) and occasionally also to age and sex.

The *reproduction of innumerable pertinent studies and tables would serve no* practical purpose for they have only a little historical interest.

All investigations reveal that in adults the average value of different measurements of the sagittal orthodiagram steadily change with height, weight, and chest circumference, they increase and decrease in the same direction as body measurements. This *indicates that different diameters of the sagittal orthodiagram reflect* the true heart size and that the various body measurements exert a similar influence on average heart size. The latter is explained by the close connections existing between diverse dimensions of a man of normal build (Dietlen) When this harmony is disturbed, strict correlation between different body measurements and those of the sagittal orthodiagram are missed but this does not necessarily mean that the heart is abnormal Thus, in people of equal weight but of different height, there is no corresponding increase of single cardiac measurements with greater height but an almost constant and, partly, even decreasing value for Tr_H, Hl, and Hfl (Dietlen). Undoubtedly in the subjects studied, only the shortest groups of individuals were normal in weight and were well proportioned while those in taller groups were somewhat underweight and ptotic and may have had vasomotor instability and poor musculature, consequently—in respect to height—the hearts of the latter were relatively small (p 102) On the other side, when individuals with a normal circulation and of the same height are arranged according to different weights, heart size does not increase in accordance with increasing weight since the fat deposits, primarily responsible for the excess weight in the heavier groups, exert only a slight influence on cardiac size. Obese individuals with normal circulation have hearts which are relatively small in relation to their weight while underweight individuals have hearts too small for their height. Only when individuals are normal in nutrition and proportions do height and weight go almost parallel The correlation in children is more flexible in respect to cardiac measurements and body measures since in the growing organism the ratio between the size of single organs and their parts is even more variable than after growth is concluded.

The close relation, repeatedly confirmed for adults of normal build, between the average value for single measurements of the sagittal orthodiagram and various body measurements at the moment seemed to provide a reliable answer to the question whether the heart was of normal size or not, but this hope soon was dissipated for the scatter of deviations of values of the sagittal orthodiagram for various body dimensions normally has a remarkable spread around the

average value. Consequently, no conclusion about the normality of heart size for the given case is possible. This is evident *from the following fact: even within a* closely drawn height and weight class, deviations of cardiac measurements spread so widely that the maxima of one class can greatly exceed the average value in the next higher class (Otten)

This can be explained *largely* by the following fact despite a fixed cardiac volume, single measurements of the sagittal orthodiagram can differ greatly depending upon the shape of the heart and its position in the thorax so that there is no measure even partly reliable for determining true cardiac size. The dependence of measurements of the sagittal orthodiagram on the position of the heart in the thorax was mentioned on page 70.

An advance in this respect was achieved by the cardiac correlation of Rautmann Actually in this procedure as well, the transverse cardiac diameter alone was considered. This imperfect definition of cardiac size was partly corrected by relating it to height, weight, and chest circumference, that is, to the three body measurements which together determine the size as well as the position of the heart in the thorax.

From approximately 1800 orthodiagrams of healthy young males obtained in the sitting position, Rautmann calculated the correlation and regression coefficients for Tr_H · height, Tr_H chest circumference, and Tr_H weight. On the basis of these calculations he tabulated Tr_H together with corrections for weight and chest circumference, (tables 1 to 3)

TABLE 1 —*Tabellar arrangement of the transverse cardiac diameter (Rautmann)*

Height cm	Weight Kg	Chest circumference cm	Tr_H cm	Height cm	Weight Kg	Chest circumference cm	Tr_H cm
150	51 0	81 0	12 8	173	67 1	86 8	13 4
151	*51 7*	*81 3*	*12 8*	*174*	*67 8*	*87 0*	13 4
152	52 4	81 5	12 9	175	68 5	87 3	13 4
153	53 1	81 8	12 9	176	69 2	87 5	13 4
154	53 8	82 0	12 9	177	69 9	87 8	13 5
155	54 5	82 3	12 9	178	70 6	88 0	13 5
156	*55 2*	*82 5*	*13 0*	*179*	*71 3*	*88 3*	*13 5*
157	55 9	82 8	13 0	180	72 0	88 5	13 5
158	56 6	83 0	13 0	181	72 7	88 8	13 6
159	57 3	83 3	13 0	182	73 4	89 0	13 6
160	58 0	83 5	13 1	183	74 1	89.3	13 6
161	58 7	83 8	13 1	184	74 8	89 5	13 6
162	59 4	84 0	13 1	185	75 5	89 8	13 7
163	60 1	84 3	13 1	186	76 2	90 0	13 7
164	60 8	84 5	13 2	187	76 9	90 3	13 7
165	61 5	84 8	13 2	188	77 6	90 5	13 7
166	62 2	85 0	13 2	189	78 3	90 8	13 8
167	62 9	85 3	13 2	190	79 0	91 0	13 8
168	63 6	85 5	13 3	191	79 7	91 3	13 8
169	64 3	85 8	13 3	192	80 4	91.5	13 8
170	65 0	86 0	13 3	193	81 1	91 8	13 9
171	65 7	86 3	13 3	194	81 8	92 0	13 9
172	66 4	86 5	13 4	195	82 5	92 3	13 9

In table 1, heights from 150 to 190 cm. are arranged for corresponding averages of weight, chest circumference, and Tr_H. If weight of chest circumference deviates from these averages, the value of Tr_H must be increased or diminished by the amount reported in the correction tables (tables 2 and 3), in this way the predicted value of

TABLE 2 —*Correction table in regard to weight (Rautmann)*

With a deviation of body weight from the average value

About	1 00– 2 25 Kg	alters the Tr_H about	0 1 cm
	2 25– 3 75		0 2
	3 75– 5 25		0 3
	5 25– 6 75		0 4
	6 75– 8 25		0 5
	8 25– 9 75		0 6
	9 75–11 25		0 7
	11 25–13 25		0 8
	13 25–14 75		0 9
	14 75–16 25		1 0
	16 25–17 75		1 1
	17 75–19 25		1 2
	19 25–20 00		1 3

TABLE 3 —*Correction in regard to chest circumference (Rautmann)*

With a deviation of chest circumference* from the average value

About	1 00– 2 25 cm	the Tr_H changes about	0 1 cm.
	2 25– 4 25		0 2
	4 25– 5 75		0 3
	5 75– 7 25		0 4
	7 25– 9 25		0 5
	9 25–10 75		0 6
	10 75–12 25		0 7
	12 25–14 25		0 8

* The chest circumference is calculated as the average value in deepest inspiration and expiration The measuring band is applied parallel to the outstretched arms just below the level of the nipples

Tr_H is obtained for the height, weight, and chest circumference of the subject From comparison of the Tr_H found with the value calculated from the three tables, it should be possible to decide whether the particular heart is normal in size or not. No deviations of Tr_H up to 0.6 cm of the predicted value should be considered unconditionally pathologic.

From an empiric formula, Hodges and Eyster calculated the normal value of Tr_H in sitting males by including height, weight, and age

$$Tr_H \text{ (mm)} = \text{age (years)} \times 0.1094 - \text{height (cm)} \times 0.1941 + \text{weight (Kg)} \times 0.8178 + 95.8625.$$

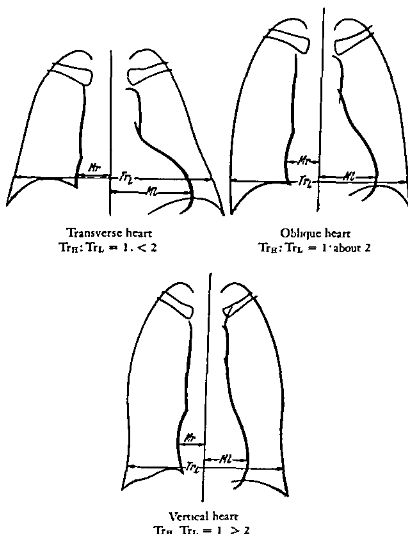
If Tr_H of the particular patient exceeds by 5 mm. the predicted value determined by this formula there is presumed with a 3:1 probability that the enlargement is pathologic

Finally, Breitmann attempted to calculate the value of Tr_H from height (L) weight (P), and chest circumference (C). By comparing Rautmann's figures, he concluded that Tr_H of the male heart is closely approximated by the formula.

$$Tr_H = \frac{1}{120} L + \frac{1}{90} P + \frac{1}{30} C + 8.3.$$

For the female heart, the coefficient employed is 7.8 instead of 8.3 (Maislich and Bobrezkaja). Values deviating more than 6 mm. above or below the value predicted by Rautmann of Tr_H are considered abnormal

The same inadequacies mentioned as impairing correlative cardiac dimensions also affect those depending upon calculations of constant numeric ratios (quotients)



Transverse heart
$Tr_H : Tr_L = 1 . < 2$

Oblique heart
$Tr_H : Tr_L = 1$: about 2

Vertical heart
Tr_H $Tr_L = 1 > 2$

Fig 42.—Groedel's heart-lung ratio Its dependence upon spatial relations in the chest and particularly on the level of the diaphragm.

between various measurements of the sagittal orthodiagram and different body measurements.

The correlation between transverse cardiac dimension and that of the thorax found widest acceptance. After Kreuzfuchs (1912) related the size of the heart to width of the thorax by calculating the relation of cardiac breadth to thoracic breadth with central projection, Th. and F. Groedel (1915) investigated this relation ortho-

diagraphically. They devised a cardiothoracic ratio, $Tr_L : Tr_H$ in which Tr_L was the greatest horizontal distance between the lateral inner borders of the ribs at the level of the diaphragmatic domes (fig. 42). This ratio is very constant (Th. and F. Groedel). In children between 3 and 10 years it amounts to 1:1.9, in men of 20 to 1:1.92 and at 30 to 1:1.95. In other words, the childhood heart is relatively broader in relation to the thorax than the heart of middle age when the ratio is approximately 1:2. Major deviations from these figures suggest enlargement or abnormal smallness of the heart (Groedel). In extensive material Hammer's average in healthy males was 1.98 with the rather large deviations of 1.8 to 2.3. On the basis of Haudek's studies, Dietlen calculated the deviations of the cardiothoracic ratio spread from 1.84 to 2.25. In children Kirsch found an average cardiothoracic ratio of 2.015 with average deviations of 1.8 to 2.2 and maximal deviations of 1.74 to 2.46. On the basis of personal studies plus those of Bamberg and Putzig and Lange and Feldmann, Kirsch assumed a progressive relative reduction of the transverse cardiac diameter in early childhood. His average values were: first months of life, 1.83, fourth to fifth months, 1.87, eighth to ninth months, 1.93; beginning of second year, 1.94; third to fourth years, 1.99. The values attained by the third year remained unaltered through the entire period of growth and even during adult life. Danzer (1919) concluded that the transverse cardiac diameter amounted to 39 to 50 per cent of the transverse diameter of the thorax with an average of 45 per cent, a value of 50 per cent was suspect and values higher than this were definitely pathologic.

The ratio of heart and thoracic breadth evidently follows general laws but in individual cases is less constant than Th. and F. Groedel originally assumed. Marked deviations from average values are common in women whose transverse cardiac diameter is often relatively large in relation to thoracic width; consequently their cardiothoracic ratios may be remarkably small in the absence of cardiac enlargement. The thorax of many females is relatively deep but narrow and consequently approaches the infantile form. Therefore cardiothoracic ratios are like those found in childhood (Groedel, Kirsch).

The value of the cardiothoracic ratio is decidedly limited owing to influence by alterations of diaphragmatic level (fig. 42). The transverse cardiac diameter (Tr_H), to a great extent, depends on the position of the heart in the thorax (p. 182). A transverse cardiac position from elevation of the diaphragm is accompanied by an increase of Tr_H, cardiac obliquity from diaphragmatic descent by a reduction. Often this shifts the cardiothoracic ratio. Even if the elevation of the diaphragm (obesity or space-occupying abdominal lesions) increases Tr_L as well as Tr_H, the latter usually increases relatively more than the former. This alters the ratio $Tr_H : Tr_L$ in the sense that a normal but transverse heart seems enlarged. On the contrary, in many ptotic individuals with a spacious thorax and a relatively wide, lower thoracic aperture, the Tr_H of the median placed and vertical heart is proportionately small to Tr_L, simulating an abnormally small organ, then, a heart actually enlarged may appear normal in size if the cardiothoracic quotient alone is considered.

Owing to this dependence of the cardiothoracic ratio on diaphragmatic position, Dietlen calculated (on horizontal orthodiagrams) individual average quotients for different cardiac positions. His value was 2 for the oblique, 2.15 for the vertical, and 1.9 for the transverse heart; for pendulous hearts the average value was 2.4.

For the female heart, the coefficient employed is 7 8 instead of 8 3 (Maislich and Bobrezkaja). Values deviating more than 6 mm. above or below the value predicted by Rautmann of Tr_H are considered abnormal

The same inadequacies mentioned as impairing correlative cardiac dimensions also affect those depending upon calculations of constant numeric ratios (quotients)

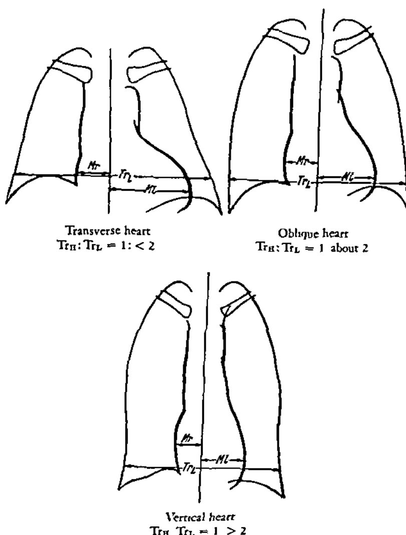


Fig. 42 —Groedel's heart-lung ratio. Its dependence upon spatial relations in the chest and particularly on the level of the diaphragm.

between various measurements of the sagittal orthodiagram and different body measurements.

The correlation between transverse cardiac dimension and that of the thorax found widest acceptance. After Kreuzfuchs (1912) related the size of the heart to width of the thorax by calculating the relation of cardiac breadth to thoracic breadth with central projection, Th and F. Groedel (1915) investigated this relation ortho-

In normal hearts the ratio of OTD: OCS should be 1·2, with maximal deviations of 1·2.3 and 1:1.8. The ratio of 1 1.8 or less should be found only with enlarged hearts.

The depth diameters a and b were considered measures of right and left ventricular size. Both a and b should be about equal to $\frac{OCS}{4}$. Increases of either beyond this value indicate enlargement of the right or left ventricle respectively.

This fairly complicated reconstruction provokes mental reservations because Fray obtained them by measuring teleroentgenograms which includes the errors associated with every central projection. A second important source of error is the dependence of the cardiothoracic ratio on the level of the diaphragm and the shape

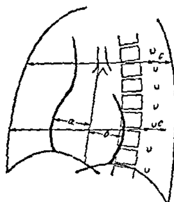


FIG 43.—Correlative determinations of cardiac size and of the two ventricles, according to W. W. Fray.

of the chest, this is not circumvented any more than in the Groedel ratio Consequently deviations of values are equally great and the new method has no advantage over Dietlen's cardiothoracic ratio or the Alstadt modification of Groedel's method

The relation between the transverse cardiac diameter and the width of the shoulders (biacromial distance) recommended by Taipale is also not essentially different, in respect to fundamentals or results, from Groedel's ratio

Somewhat better are correlation quotients calculated from various cardiac and body measurements which define the body more precisely than does the transverse diameter of the lungs In this connection the studies of Moritz and his pupils deserve emphasis

Considering that individual differences in development of the fat pads widely parallel body weight without exerting noteworthy influence on heart size within wide limits, Moritz substitutes the ideal weight for the actual body weight and relates this to cardiac surface (Hfl) according to the formula $\frac{\text{cardiac surface}}{\text{ideal weight}}$, ideal weight is calculated from Bornhardt's formula, $\frac{\text{height} \times \text{chest circumference}}{240}$ The quotient $\frac{\text{cardiac surface}}{\text{ideal weight}}$ thus obtained should be very constant (Moritz) and should average 20 1.

For the typical transverse female heart the average value was 1 92, for the vertical 2 13 and for all female hearts 1.96.

On the basis of extensive personal observations Ludwig denied that the cardiothoracic ratio was useful for measuring cardiac size

Altstadt made a valuable suggestion in individuals with an elevated diaphragm the transverse cardiac position should be abolished by deep inspiration, the transverse diameter of the heart is determined at a depth of inspiration when the angle of cardiac inclination is 45 degrees ("normal situation of the heart," Haudek) This value is related to the transverse diameter of the thorax, not in inspiration but, as usual, in average expiration This procedure certainly yields more comparable and very useful cardiothoracic ratios Nevertheless the person under investigation should not involuntarily strain to maintain deep inspiration since this, as is well known, reduces the size of the heart (p 108)

Recognizing that the transverse diameter of the heart defines cardiac size very imperfectly, Bamberg and Putzig calculated the ratio of the lung transversal Tr_L and the "average cardiac diameter" which is the arithmetic mean of Tr_H, L, and Br. With teleroentgenograms in nurslings this ratio is 1 85 to 2. Von Bernuth found a value of 1.93 to 2 5 (average of 2 17) for the same ratio in children over 1 year old, that is, the breadth of deviations was very large

H Zondek endeavoured to find the normal cardiac size more precisely by retaining the transverse cardiac diameter as a relative measure while substituting a "thoracic index" for the transverse diameter of the lungs The index was calculated *from the thoracic height (b) and width (a) by the formula 2a-b He arranged the* various "thoracic indices" according to average values, found empirically, of the transverse cardiac diameter Deviations from the predicted values should permit one *to recognize a heart as too large or too small*

In recent American literature examination in the left anterior oblique position has found special favor since with rotation to the right of about 40 degrees the ven*tricular septum runs approximately in the path of the rays so that each half of the* heart projects about equally to right and left Fray measured the transverse diameter of the cardiac shadow and of the thorax in this position and thought he could express numerically, enlargement of the entire heart or of either half The patient is rotated to the right until the cardiac shadow is smallest With a normal position of the diaphragm this is about 40 degrees, with descent or elevation less or more than 40 degrees, respectively The course of the ventricular septum is constructed as follows (fig. 43) the horizontal distance from the right anterior bony chest framework to the right tracheobronchial angle is measured and the line continued at the same level posteriorly. This line passes over the ventral limit of the joint of the left costotransverse process posteriorly for the distance C In the same position, the "thoracic transversal" (OCS) is constructed by drawing a line connecting the right anterior chest wall and the ventral limit of the left costotransverse process joint at the cardiac level (?) The thoracic transversal *is prolonged the length C and at the* middle, the line is joined to the right tracheobronchial angle by a straight line which indicates the location of the ventricular septum From this straight line the longest plumb lines are dropped *to the anterior* (a) and *posterior* (b) limits of the cardiac shadow. The sum of a + b gives the transversal of the cardiac shadow (OTD)

9.5) whereby values below 8 indicate the heart is too small and values over 11, too large. For erect women the quotient is smaller, it amounts to 7.31 to 10.88 and averages 8.88 whereby values under 7 should indicate that the heart is too small and values over 11 that it is too large. Kahlstorf stressed that hearts too large or too small according to this calculation are encountered without any clinical signs of circulatory disturbance or cardiac impairment. In our experience a reliable conception of the normality of cardiac size is obtained by relating the ideal weight calculated by the Bornhardt method (p. 85) instead of the actual weight to cardiac volume.

All these ratios have a scattering of deviations about the average which is so broad that they are poorly adapted for definite statements about the normality of cardiac size in a particular case. This is inevitable from the fact that one or another body measurement is considered at the moment in the various formulas although in no instance does it alone determine cardiac size nor does it change in absolute proportion. This can be deduced even from the first studies of Dietlen and others.

Recently on the basis of detailed mathematic treatment and extensive personal investigations, Ludwig examined the question of correlative determinations of cardiac size. As the outcome of his studies, he presents table 4 from which the most probable prediction (± 11 per cent) of cardiac volume can be determined from a given weight in kilograms (g), a given thoracic breadth (Tr_L at the height of the right diaphragmatic dome) in centimeters (b) and a given thoracic depth (at the same level) in centimeters (t) by the addition of three numbers.

TABLE 4.—*Predicted Cardiac Volume from Body Weight and Breadth and Depth of Thorax (Ludwig)*

Weight		Breadth of thorax		Depth of thorax	
40	217	20	−41	7	128
45	244	21	−24	8	146
50	272	22	− 7	9	165
55	299	23	+10	10	183
60	326	24	27	11	201
65	353	25	44	12	220
70	380	26	61	13	238
75	407	27	78	14	256
80	435	28	94	15	275
85	462	29	111	16	293
90	489	30	128	17	311
95	516	31	145	18	329
100	543	32	162	19	348

For interpolation one adds 5 cc. for every 1 Kg. and 2 cc. for every 1 mm. of thoracic breadth or depth.

In a given case, a cardiac volume determined by the Rohrer-Kahlstorf method which exceeds the predicted value by +11 per cent determined from table 4 means cardiac enlargement with a probability of 84 per cent.

When roentgenologic determination of cardiac volume is impossible or inexact owing to difficult demarcation of the cardiac apex, Ludwig recommends as a relative

On the basis of Hammer's studies, Moritz later thought it advantageous to relate the cardiac surface (Hfl) or rectangle (Hr) to the body rectangle, height × Tr_L according to the formula $\frac{\text{cardiac surface} \times 10}{\text{body rectangle}}$ or $\frac{\text{cardiac rectangle} \times 10}{\text{body rectangle}}$. Average values for these quotients for the erect position are 226 or 300 (Hammer) and for the dorsal recumbent position 270 or 356 The values in individual cases were expressed as percentages of these predicted amounts so that deviations ± 10 per cent were regarded as normal and only greater ones were considered indicative of abnormal cardiac smallness or enlargement.

With cardiac teleroentgenograms and by employing the same dimensions, v. Bernuth calculated the correlative cardiac size in children from the age of 1 year to puberty according to the formula, $\frac{\text{body rectangle}}{\text{cardiac surface}}$. In the erect position these quotients average 39.3 with deviations of 31 to 47 which is regarded as normal; for $\frac{\text{body rectangle}}{\text{cardiac rectangle}}$ the average is 29.3 with maximal deviations of 23 to 35.

Subsequently, Moritz concluded that the use of his body rectangle or Hammer's had no advantage as a correlate of cardiac size over height and was even inferior. Accordingly he recommended height as the sole correlate He expressed the correlations of height (Kl) and various cardiac measurements as "body-cardiac length," $\frac{\text{Hr}}{\text{Kl}}$, and "body-cardiac surface," $\frac{\text{Hfl}}{\text{Kl}}$. In 100 males and females with healthy hearts, he calculated the average of these quotients to obtain normal values A glance at his tables shows, however, that the hearts of noncardiac patients show great deviations in average values so that no insight is gained in respect to the normality of cardiac size.

Definite progress was achieved when cardiac volume, roentgenologically determined, was considered. At an early stage, Geigel calculated the volume of the heart (p 76) in relation to the nude weight (G) by the formula $V = F^{3/2} \times \frac{4}{3\sqrt{\pi}}$ through the cardiac quotient $HQ = \frac{V}{G}$. By disregarding the absolute value, the constant $\frac{4}{3\sqrt{\pi}}$ was eliminated and the reduced cardiac quotient $rHQ = \frac{F^{3/2}}{G}$ was ascertained. For normal individuals this amounts to 14 to 22. Values under 14 indicate the heart is too small and those over 22 that it is too large. Similarly Hecht calculated the relative cardiac size of children according to the formula $\frac{\text{cardiac surface}}{\text{weight}^{2/3}}$ and obtained an average value of 737 with considerable deviations above and below, these he explained in part by large differences in the fat deposits of children.

Kahlstorf advised relating the cardiac volume obtained by him and Rohrer to weight. He calculated the "cardiac quotient" $\frac{\text{cardiac volume in cc}}{\text{nude weight in Kg.}}$ and finds in normal adults, neither obese nor underweight, that cardiac volume is a linear function of body weight. The quotient (Kahlstorf) for erect men is 8.15 to 10.9 (average

relatively heavy and therefore larger, but by the second year it reaches a weight ratio which remains fairly constant in later years (Roessle and Roulet).

With increasing age of the child, the average of various cardiac measurements progressively rises corresponding to the greater mass of the child's body (Veith, v Bernuth, Otten, Lehmkuhl, Kirsch, Dietlen and Schall) According to v. Bernuth in all groups of children, sex differences are noted, for girls in the prepuberal period have, at first, smaller cardiac measurements than boys. At the time of puberal growth which usually starts earlier in girls, measurements of the male heart remain behind those of girls but later this difference disappears Here again the difference is conditioned by discrepancies in body development and is not sex-specific

The roentgenologic studies of Veith, Th. Groedel, Bamberg and Putzig, v. Bernuth, and Kirsch, as well as of Dietlen and Schall agree that even in the child average cardiac measurements increase continually with height but without an appreciable sex difference. The particularly wide breadth of variations in cardiac measurements of children may be explained—as previously indicated—by the fact that in the developing organism, correlations between cardiac size and the different body dimensions are not as fixed as in adults Thus, it often happens (Rauchfuss) that there is a temporary disproportion between cardiac size (also, in the width of the vascular system) and other developments of the body. Particularly noteworthy in this respect is the scattering of correlative cardiac measurements with respect to body weight since the local accumulation of fat in children varies remarkably without essentially influencing cardiac size (Bamberg, Putzig).

The heart of the aging organism takes a special position. The heart increases in weight somewhat near the turn of the fourth decade into the fifth and then decreases at more advanced ages (Roessle). This added weight corresponds to a slight but distinct increase in all cardiac measurements at this period (Dietlen) Dietlen stresses that this enlargement involves primarily the mid-left distance (Ml), for increasing demands are placed on the heart through aging of the peripheral circulation, moreover, the development of emphysema frequently leads to ventricular hypertrophy and dilatation.

Atrophy and diminution of cardiac size in old age has been established anatomically (Roessle and Roulet). It is found, however, only when the body mass, skeletal musculature, and fat deposits succumb to senile atrophy. Muscular, well nourished individuals maintain normal cardiac measurements into advanced age. In general, dimensional alterations of the heart are only slightly influenced by aging

Among the innumerable methods for determining normal cardiac size, only a few are selected for mention in this place The constant increase in their number, by itself, suggests that all are unsatisfactory although considerable progress has been made steadily After all, even at present, calculations of the normal still yield such wide deviations in respect to correlation, and variations from the average are so great, that it is impossible to decide whether the heart should be considered normal, too large, or too small for the particular carrier These discrepancies occur not only with methods which correlate one or another linear measurement of the sagittal orthodiagram with this or that body measurement but also with the best correlative cardiac measurements available. The reasons are as follows

1. No calculation can place a numeric value upon some important factors par-

TABLE 5 —*Calculation of cardiac volume from* $T + T_{90h}$

$T + T_{90h}$	Volume in cc	$T + T_{90h}$	Volume in cc
17	320	24	740
18	380	25	800
19	440	26	860
20	500	27	920
21	560	28	980
22	620	29	1040
23	680	30	1100

For interpolation one adds 6 cc for each 0 1 cm.

volumetric measure the sum of the cardiac transversal (T) and the greatest horizontal depth diameter of the heart (T_{90h}) and reads the absolute volume from table 5.

The cardiac volume of the case under investigation as shown by the table is compared to the predicted volume in table 4

After further studies Ludwig concluded that body weight in women (g) is the decisive factor for cardiac volume and that one can calculate this predicted value by the simple formula $V = 5\ g + 150\ (\pm 12$ per cent)

By means of the various correlative determinations of heart size reviewed above Dietlen, Claytor and Merill, Francke, Groedel, Otten, and Ludwig studied the influence of sex on heart size In agreement with anatomic studies (W Müller, Roessle and Roulet), all authors found the average value was smaller in the adult female than in the male. Ludwig's average showed the female heart about 20 per cent smaller than that of males This sex difference can undoubtedly be explained by the fact that ordinarily males work harder physically and their skeletal muscles are more powerfully developed than those of women, fat deposits are relatively more developed in women than in men and exert only a slight influence on cardiac size (Dietlen). At this time, it may be recalled that males with light occupations and weaklings have smaller hearts than those engaged in heavy labor and sports The heart of a woman who works daily and has well developed muscles equals that of a male of the same weight and same muscular development.

During pregnancy the heart enlarges slightly in proportion to the increase of weight (Binhold), this is not a dilatation or hypertrophy which exceeds the values which might be anticipated by the larger body measurements Long ago Dietlen stated that the cardiac enlargement repeatedly reported on the basis of clinical and roentgenologic studies was greatly overemphasized and, for the most part, illusory owing to elevation of the heart and its transverse position (Klaften and Palugyay, Maislich and Bobrezkaja)

Many studies have been made on the influence of age on cardiac size. In middle age no effect has been shown. Special situations occur, however, in growth and in old age.

Anatomic studies show that the growth of the child heart is not uniform but is accelerated during the first year of life and at puberty. Although precisely at these

Measurement reveals that not only Tr_H diminishes in the erect posture but in 60 per cent of the cases cardiac breadth (Hbr) decreases while length (H) may remain the same, decrease or occasionally increase. Accordingly, the heart occasionally seems more slender in standing than in recumbent individuals.

As a rule cardiac surface area (Hfl) is smaller and this reduction (Moritz) may amount to 25 per cent or more Since the reduction is not limited solely to the sagittal projection of the heart but also affects its depth dimension, it is evident that the heart normally becomes smaller in the erect position (Moritz) F. M. Groedel and others thought the reduction an illusion produced by a change of cardiac shape and position as the result of altered spatial conditions They based this upon the observation that orthostatic reduction of the sagittal image tends to be most striking when the

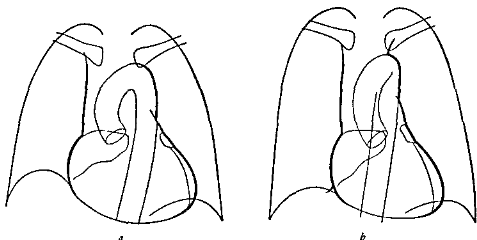


FIG 44—Alteration of the shape, size, and position of cardiovascular shadow on change from recumbent (*a*) to erect (*b*) position

diaphragm descends markedly in the erect individual, then the heart also experiences a very striking change in form and position This objection was, however, refuted by the fact that this reduction appears not only in the sagittal view but also in the frontal and, indeed, in both so decidedly that a particularly marked orthostatic reduction of heart size must occur in these cases

The amount of orthostatic reduction of cardiac size can vary decidedly It tends to be greater in normal hearts than in moderately enlarged ones, when the heart is greatly enlarged it is often missed entirely (Dietlen) This rule has exceptions, for hearts dilated as the result of infectious diseases may show very striking orthostatic reductions even though they are greatly enlarged (Dietlen, Zdansky).

The degree of orthostatic reduction of cardiac size is determined by the following factors (Dietlen)

1. Myocardial thickness. Thin hearts offer less resistance to alterations of their size than thick-walled ones This is one reason why hypertrophied hearts, though markedly dilated, usually show trivial or no orthostatic reduction As a matter of fact, Dietlen proposed this sign precisely for the differential diagnosis of cardiac hypertrophy and dilatation

ticipating in cardiac size, on the actual strength of the skeletal muscles, the type and extent of their activity, for example.

2 Even if one succeeded, despite considerable individual variations in cardiac size as determined roentgenologically, obviously cardiac size is determined not only by the status of its muscle but also by its blood content which is subject to decided fluctuations.

Gerhardt very properly warned against the "pseudo-accuracy of numbers" and Otten believes that alterations in the shape of the cardiac shadow are more important in recognizing morbid conditions than deviations from statistical predictions of average values, this statement contains many elements of truth

Although roentgenologic measurements usually are not conclusive in determining the normality of cardiac size in individual and borderline cases, nevertheless they have great practical value They agree remarkably with average predicted values so that it is possible to investigate, in a large series, and to express mathematically, the regular dependence of cardiac size on various intrinsic and extrinsic factors This alone justifies the countless efforts which have been made to obtain correlative cardiac measurements

Even in single cases absolute and correlative cardiac measurements are not worthless since with their help alterations of cardiac size and its correlations can be followed in the individual subject when the changes appear under the influence of definite measurable or even arbitrarily arranged conditions, among these are work and certain athletic activities. This may have decided importance in decisions about the heart

VII. The Heart and Position of the Body

As already mentioned (p 78) roentgenologic measurements of the heart are systematically recorded in the supine position This standard (Moritz, Dietlen) is based upon the knowledge that cardiac size and shape are profoundly influenced by posture To Moritz's demonstration in 1904 nothing fundamental has been added about statically conditioned positional changes of size and shape of the cardiac shadow.

When one rises from the horizontal to the erect position, the diaphragm descends and the heart, resting on it, follows the downward movement (fig 44a and b) Thus, the heart becomes more vertical since the right border can change its position but little while the left border with the apex definitely swings median and down Owing to simultaneous rotation of the heart to the right, the left atrium rotates to the left and somewhat ventrad so that it may project as a flat bulge in the cardiac waist (Schwarz).

The supracardiac vascular complex moves caudad with the heart as far as its connection with the cervical fascia and the left main bronchus permit Since these connections limit the descent, the aortic arch is subjected to traction by the heart so that its limbs extend less to the right and left In this way the entire vascular band becomes smaller and longer so that the aortic knob flattens and moves caudad from the clavicular shadows

the abnormal reduction of tonus in the peripheral vessels and skeletal muscles. Peripheral vasomotor weakness can be constitutionally conditioned as in many asthenics, neurotics, and in overgrown youths (Kraus, Schiff, Dietlen, Pal), it can also be released by central, pituitary, inter-renal, or peripheral reflex mechanisms as exemplified by tabes or in hypophyseal cachexia, Addison's disease, and infectious-toxic states. Very often reduced vascular tonus is associated with lowered tonus of

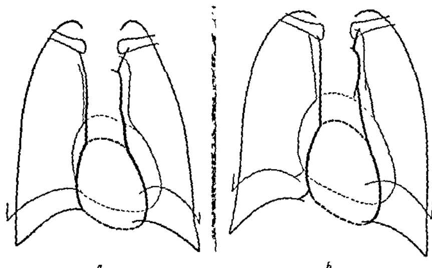


Fig 45 —Marked orthostatic reduction of heart size (*a*) Male, 32 years old, with multiple sclerosis (*b*) Asthenic individual, 30 years old, with pulmonary tuberculosis

	Case *a*		Case *b*	
	Lying	Standing	Lying	Standing
Cardiac volume in cc, after Rohrer	537	337	610	463
Circulating blood volume in liters	5 2	3 8	5 0	4 4
Blood pressure in mm Hg	105/65	75/0	110/60	130/100*
Diameter of aortic arch (Kreuzfuchs) in mm	24	20	23	23†

* Despite diminished circulating blood volume, blood pressure rose on standing
† Aortic diameter was not recorded in erect position.
Heavy line vertical orthodiagram, thin line horizontal orthodiagram

skeletal muscles (Henderson, Beiglebock) and this may greatly promote storage of blood in the abdomen and in dependent parts.

Finally, when one considers that the heart is often definitely thin-walled and relaxed in precisely these conditions, one understands that the diminished return of blood may be adjusted with special ease by a corresponding reduction of cardiac size (Dietlen) Moreover, a considerable increase of heart rate, orthostatically conditioned, may contribute to the reduction of cardiac volume.

Moritz attempted to define the orthostatic alterations of cardiac size and shape

2. Myocardial tonus. Although this function of the heart muscle cannot be measured exactly, it plays a role in the stability of cardiac size and shape. If one assumes with Dietlen that a thick (hypertrophic) heart muscle, *ceteris paribus,* also has higher tonus, the lesser orthostatic reduction of the hypertrophic heart can be conceived as an expression of higher tonus.

3. Heart rate Acceleration takes place predominantly by shortening of the diastolic filling phase and consequently reduces cardiac size (Moritz, Dietlen, Scherf and Zdansky, Meek) As is well known, standing frequently actually increases cardiac rate although normally this is not very significant and moves within limits inadequate to exert any essential effect on diastolic cardiac size. Nevertheless, orthostatic tachycardia occasionally attains rates that may reduce cardiac size

4 Flow of blood into the heart. Normally this is less in the erect posture than in the recumbent since on standing some blood is retained in the splanchnic field and in dependent parts of the body (Hill, Lindhard, Moritz, Laurell, and others). The amount of blood so stored may suffice to diminish the volume of circulating blood significantly

Since so many factors participate in the orthostatic reduction of cardiac size it is understandable that the amount of reduction may vary greatly and that there must be cases in which it may become extremely important owing to special situations or by summation of several factors Thus, in individuals healthy from the standpoint of circulation, Zdansky found cardiac volume, calculated by the Rohrer method, occasionally 200 cc less in the erect position than in the recumbent These results are obtained particularly in ptotic-asthenic individuals, in "ill-developed giants" (Kraus), in many overgrown youths, and even in small, thin, delicately built people, especially women

Extreme orthostatic reduction of cardiac size occurs relatively often in multiple sclerosis, tabes, thyrotoxicosis, emaciation and cachexia as well as in acute cardiac dilatation due to toxic-infectious states (fig. 45a and b). In all these conditions, when the patient stands, the strikingly small, median placed heart of more or less mitral configuration shows fast, lively pulsations, it becomes normal in size and shape and displays quiet, powerful pulsations immediately when the patient reclines

In all these conditions which stand partly within normal limits and partly in the domain of pathology, clinical examination of the erect patient often reveals signs of arterial anemia like vertigo, pallor, headache, fatigueability, tachycardia, fall of blood pressure, and tendency to collapse Corresponding to these observations, Laurell and Zdansky demonstrated a considerable reduction of circulating blood volume when these patients stood

This decrease of circulating blood volume has different causes In his classical studies Wenckebach first showed that the low, flat diaphragm and the relaxed abdominal wall of the ptotic-asthenic individual supported the circulation poorly and could create such unfavorable conditions for venous return that relatively large amounts of blood were stored in the abdominal viscera and failed to reach the heart Then, upward displacement of the diaphragm by pressure exerted from below on the abdominal wall (Glénard's maneuver), often sufficed in these cases to bring cardiac size to normal with disappearance of all signs of arterial anemia.

More important for the occurrence of defective return of blood to the heart is

discussed the patient should breathe regularly and quietly and should not, at least for twenty-four hours in advance, undertake any exertion inappropriate for the study.

Whereas investigation in the erect position is indispensible for exact analysis of the cardiovascular complex, the recumbent posture is equally important for the most reliable determinations of cardiac size and of actual aortic diameter. More specifically, the horizontal position excels all others for establishing normal values of heart size since the results are most constant. Hammer found that the variations of Tr_{H} in the same individual were twice as large when standing as when sitting and this need not depend upon the fact that sitting subjects are quieter than standing ones, as he thought, rather fluctuations of cardiac inflow are much greater when standing than when sitting

With the obvious superiority of the horizontal position it seems debatable whether the determination of normal values is, generally speaking, necessary or even justified on upright patients.

This situation exists because large series balance irregularities which emerge from the intrusion of static influences. Otherwise it is incomprehensible that correlative determinations of cardiac size and the investigation of the influence of different physical effects on this size, with few exceptions, reached similar conclusions irrespective of whether they were undertaken on standing, sitting, or recumbent subjects. Nevertheless, this situation prevails only when the series is large, with limited studies all determinations should be made on recumbent subjects despite any advantage offered by the erect position

Very often it may be useful to determine cardiac size with the patient standing as well as recumbent (Moritz, Dietlen, Zdansky) because a marked orthostatic reduction of cardiac size and altered shape may have great practical and theoretic interest.

VIII. Influence of Diaphragmatic Level on the Position, Shape, and Size of the Heart

About one third of the normal oblique heart lies in the right half of the thorax and two thirds in the left, the long axis runs from the right-posterior-above to the left-anterior-below The diaphragmatic aspect of the heart rests on the planum cardiacum of the diaphragm which descends like the long axis of the heart, although less abruptly, to the left and anteriorly Thus, the heart lies so to speak, in a groove formed by the diaphragmatic part of the pericardium which is fixed to the diaphragm, and by the sternocostal part of the pericardium which is adherent to the anterior chest wall The active heart can rotate as well as glide in this groove Naturally mobility is limited since the heart is enclosed on all sides by the fixed pericardium by virtue of which it is bound to adjacent organs (lungs, diaphragm, trachea, cervical fascia) through the entering and departing vessels, moreover the heart is firmly fixed near its right posterior border along an almost perpendicular line running between the venae cavae (caval axis) Finally, the mediastinal connective tissue and mediastinal pleura, as well as the elastic pull of the lungs, very effectively aid in stabilizing the position of the heart

The diaphragm has great significance in determining the position of the heart

numerically by determining "cardiac slenderness," $\frac{Hl}{Hbr}$, in the erect and recumbent positions as well as orthostatic "reduction of cardiac length," $\frac{Hl\ recumbent}{Hl\ standing}$ "reduction of cardiac breadth," $\frac{Hbr\ recumbent}{Hbr\ erect}$ and "reduction of the cardiac rectangle," $\frac{Hr\ recumbent}{Hr\ erect}$

He determined these ratios for 100 males and females, healthy from the standpoint of circulation and demonstrated that average cardiac slenderness was practically the same for both sexes and that the ratio, on transition from recumbency to standing, was unaltered or changed insignificantly in both sexes Furthermore, in males as well as females cardiac length, breadth, and the rectangle diminished on standing and the average reduction was greater in men than in women

Moritz attempted to employ these ratios for a numeric conception of anatomic alterations of the myocardium

As intimated above, the orthostatic reduction in size involves not only the cardiac shadow but also the vascular band The narrowing of the vascular band depends primarily upon the previously mentioned stretching of the aortic loop, in other words, at first the transformation depends upon the altered spatial situation In patients with considerable orthostatic reduction of cardiac size, there is an additional essential factor, namely, orthostatic narrowing of the aorta as comparative measurements of its diameter in the recumbent and erect posture show (Zdansky) Like the reduction of cardiac size, this orthostatic narrowing of the aorta results from its decreased filling Undoubtedly, reduction of blood pressure also plays a role and must be considered in measurements of the aorta (p 372)

The same statement probably is also valid for the pulmonary artery, although apparently there are no studies on this score Certainly diminished filling of the pulmonary artery participates in the orthostatic reduction of the vascular band With marked orthostatic reduction of heart size, Laurell saw the vascular lung markings become less prominent, particularly in the upper fields when patients were erect and he ascribed this to orthostatic reduction of pulmonary vessel contents

These remarks indicate that orthostatic reduction of the cardiovascular shadow is produced partly by transformation and displacement of the heart and great vessels and partly by actual reduction of cardiac size and narrowing of the great vessels These reductions in volume, differing vastly in extent, always result from a diminished blood content, the cause of which is less cardiac than extracardiac (central and peripheral vascular regulation, diaphragmatic function, respiration, tonus of skeletal muscles, and so forth) Since diverse factors regulating the flow of blood into the heart fluctuate greatly during life, indeed from minute to minute, obviously the amount of orthostatic reduction of cardiac size can change unpredictably in a given individual.

Consequently, it is necessary to stabilize these factors as far as possible and to exclude fluctuations if numerical determinations of cardiac size and aortic diameter are undertaken in respect to their normality. This objective is attained best with measurements obtained in the recumbent position (Moritz), for reasons still to be

The heart yields to the diaphragm pressing from below predominantly by shift or rotation of its moveable parts and chiefly by compression and deformation of relatively fixed parts. Since the apical portion of the ventricular cone, directed to the left-anterior-below, is the most mobile part, while the cardiac base, lying above on the right, is relatively fixed, elevation of the diaphragm causes the heart to rotate so that the ventricular cone turns to the left, up, and forward while the base and right border change relatively little Usually the forward displacement of the apex is slight and possible only to the extent that the a-p diameter of the thorax increases with elevation of the diaphragm. With the swing just described, the heart rotates around its long axis to the left so that its anterior surface turns to the left.

The rise and shift of the apex and the left border of the heart to the left lengthens the mid-left distance (Ml) On the other hand the mid-right distance (Mr) changes but little since the right border lies near the midline and is fixed along the caval axis, therefore, the right cardiac border really changes its shape only by more

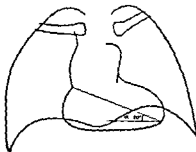


FIG 48 —Upward displacement and transverse position of the heart from elevation of the diaphragm Woman, 54 years old, with megacolon By interposition of the gas-filled colon between the liver and diaphragm, the diaphragmatic border of the heart is entirely visible The angle of cardiac inclination α = 20 degrees

marked rounding. This unequal increase of Ml and Mr shifts the ratio of Mr Ml in the direction of 1 $>$ 2 (fig 39)

The swing of the cardiac apex just described, with relatively unaltered position of the cardiac base, diminishes the angle of cardiac inclination (figs 39a and 48). The reduction of this angle is regarded as a criterion of diaphragmatic elevation when no other reason for the transverse position (elongation of the aorta, space-compressing mass of the upper mediastinum like a retrosternal goiter, or large aortic aneurysm) is demonstrable Naturally there is no parallelism between the amount of diaphragmatic elevation and reduction of the angle of inclination, that is, of the transverse position of the heart (Klaften and Palugyay) since the central tendon often changes less than the domes of the diaphragm

Since the left ventricular arc bulges more to the left when the heart becomes transverse, as a rule the cardiac waist is deeper This gives the cardiac shadow an aortic configuration and may simulate left ventricular enlargement. This is one reason why so often cardiac enlargement and hypertrophy are erroneously assumed to exist in pregnancy (Dietlen) In the oblique and lateral positions, the posterior cardiac border extends farther backward so that the retrocardiac space seems narrow and the esophagus locally experiences a backward retrocardiac indentation (fig

because it serves as a support Owing to this important function, the diaphragm demands special attention The other attachments of the heart are individually rather constant and vary comparatively little but this does not hold for the diaphragm. With each respiration and every change of position, this support changes its level decidedly and often extreme variations result in certain individuals.

Every change of diaphragmatic level alters the position and shape of the heart, naturally this is limited by other attachments and is directed into certain paths. Moreover, it is essentially affected by the size and the plasticity of the heart and great vessels. Consequently, the position of the diaphragm must receive careful consideration in every roentgen examination of the heart.

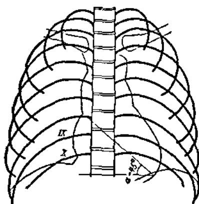


Fig 46

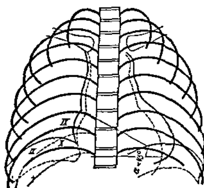


Fig 47

Fig 46 —Normal position of diaphragm The angle of inclination α = 45 degrees The dome of the diaphragm is under the ninth rib

Fig 47 —Moderate elevation of diaphragm The angle of cardiac inclination $\alpha < 45$ degrees Since the lower ribs are elevated by enlargement of lower thoracic aperture, the reciprocal relations between the domes of the diaphragm and the lower ribs is not altered to the extent that one might expect with elevation of the diaphragm Consequently the ninth rib is projected above the dome of the diaphragm despite diaphragmatic elevation (Dotted line the cardiovascular shadow as well as the ninth and tenth ribs with normal position of the diaphragm)

Normally, when the erect patient breathes quietly, the domes of the diaphragm are located at the ninth posterior intercostal spaces with the right dome a few millimeters higher than the left (Wenckebach), the upper border of the tenth rib tends to be just visible in the right cardiodiaphragmatic angle (fig 46). With inspiratory descent of the diaphragm, more of the tenth rib may appear. In the horizontal recumbent position, on the contrary, the ninth rib vanishes behind the higher contour of the diaphragm

Greater diaphragmatic elevation like that observed in the last months of pregnancy, in obesity, abdominal tumors, and ascites may be striking since the dome is at the height of the eighth posterior intercostal space or even higher. Slight elevation is not always as conspicuous and need not essentially change the relation of the dome to rib segments. This happens when the elevation of the diaphragm and widening of the lower chest aperture rotates the ribs upward at the costovertebral joints Through

relatively deep so that a horizontal section resembles a circle (Naumann, Dietlen) This produces another orientation of the heart in the chest Owing to the relatively larger depth diameter of the chest with simultaneous elevation of the diaphragm, the long axis of the heart falls more in a sagittal plane and the slope from behind and above to in front and below is less steep (fig 49) Accordingly, with sagittal passage of the ray, one sees a projectional shortening of the heart from below which contributes to disappearance of the cardiac waist. In appropriate cases, by inclining the upper body forward (fig. 51a and b), the cardiac waist actually becomes more pronounced again (Zdansky).

These explanations are not, however, applicable to all cases of mitral configuration in women for this form is not rare with normal position of the diaphragm

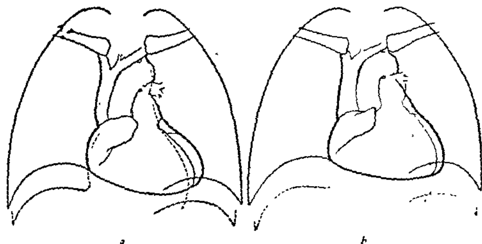


Fig. 50.—Alterations of the cardiovascular shadow from elevation of the diaphragm (*a*) Adult type Deformation predominantly from transverse position of the heart (*b*) Infantile type Deformation predominantly from compression of the heart and vessels (. normal level of diaphragm, ——— elevation of diaphragm)

and normal shape of the thorax. Therefore, many consider it a constitutional peculiarity of cardiac shape.

Elevation of one half of the diaphragm also changes cardiac position and shape in a typical manner Elevation of the right leaf of the diaphragm displaces the cardiac shadow to the left and flattens the cardiac waist to make mitral configuration more or less distinct. The latter is ascribed to lifting of the right heart and rotation to the left causing outward rotation of the pulmonary artery to the left Elevation of the left half of the diaphragm leads to a transverse position of the heart by lifting the apex and consequently to aortic configuration or displacement of the entire heart to the right, then, the heart appears median or extends more to the right than to the left, this is called dextroposition (fig. 52).

If the elevation of the diaphragm depends upon hemiparesis of the diaphragm, with paradoxic inspiratory ascent of the muscle, one observes inspiratory shift

22) like that often seen with left atrial enlargement. Later on, further reference will be made to this point. All these changes of cardiac shape vanish if deep inspiration restores the "normal situation" of the heart (Haudek), that is, it enlarges the angle of inclination to about 45 degrees.

With ascent of the diaphragm, the nadirs of the aortic loop (the aortic root and portal of the aorta through the diaphragm) are elevated, since the vertex of the loop can move upward only to a limited extent, the ascending and descending limbs of the aorta separate to the right and left respectively This widens the vascular band, accentuates protrusion of the aortic knob, and exposes more of the descending aorta in the cardiac waist The net result is further accentuation of the impression of aortic configuration and perhaps of some aortic dilatation.

Elevation of the diaphragm does not always result in aortic configuration, rather the opposite, a mitral configuration so to speak, may occur This is common in

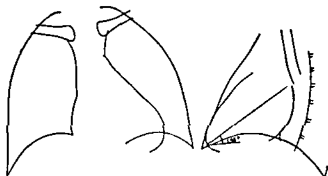


FIG 49 —"Female heart" In this case the mitral shape of the heart is certainly produced, at least partially, by the decided increase of the depth diameter of the chest and the resultant marked inclination of the heart in the dorsoventral direction The inclination of the diagonal diameter of the frontal orthodiagram (Assmann) amounts to less than 45 degrees.

childhood (fig. 36) and in adult females (fig 49), particularly in the late months of pregnancy Perhaps the mitral configuration in these cases depends, at least in part, upon pliability of the heart and vessel walls as well as of the connective tissue apparatus, peculiar to the youthful organism and to many women throughout life. Under these conditions the heart may be pushed up and flattened by the diaphragm pressing from below, then, thin-walled vessels embedded in yielding mediastinal tissue are able to spread laterally Now, the cardiac and vascular shadows appear broader, low, imperfectly separated and less differentiated The cardiac waist is filled more or less completely and the pulmonary arc can even protrude slightly (Reyher, Bamberg and Putzig) This type of deformation which is caused by [illegible] be called the infantile type (fig. [illegible] latter, deformation takes place [illegible]

Apart from these prerequisites in the tissues for the occurrence of mitral configuration of the child and "female heart," the shape of the thorax may also exert a definite effect. While the transverse diameter of the chest is essentially larger than the depth diameter in adults, the thorax of a child and of many women is small and

relatively deep so that a horizontal section resembles a circle (Naumann, Dietlen) This produces another orientation of the heart in the chest Owing to the relatively larger depth diameter of the chest with simultaneous elevation of the diaphragm, the long axis of the heart falls more in a sagittal plane and the slope from behind and above to in front and below is less steep (fig 49). Accordingly, with sagittal passage of the ray, one sees a projectional shortening of the heart from below which contributes to disappearance of the cardiac waist. In appropriate cases, by inclining the upper body forward (fig. 51a and b), the cardiac waist actually becomes more pronounced again (Zdansky).

These explanations are not, however, applicable to all cases of mitral configuration in women for this form is not rare with normal position of the diaphragm

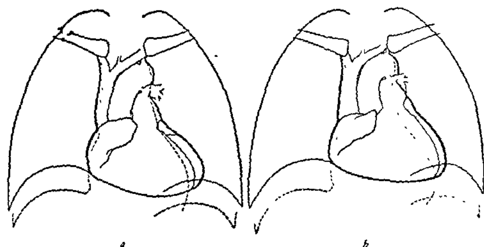


FIG 50.—Alterations of the cardiovascular shadow from elevation of the diaphragm (*a*) Adult type Deformation predominantly from transverse position of the heart (*b*) Infantile type Deformation predominantly from compression of the heart and vessels (. . . .normal level of diaphragm, ———elevation of diaphragm)

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If the elevation of the diaphragm depends upon hemiparesis of the diaphragm, with paradoxic inspiratory ascent of the muscle, one observes inspiratory shift (wandering) of the cardiac shadow to the side with normal function

Descent of the diaphragm is characterized by flattening of the dome and diminution of its respiratory excursion. Even minor grades are made evident by the appear-

ance of the tenth or eleventh rib above the dome (fig. 53). The twelfth rib becomes visible only when the diaphragm is extremely flat and with coexisting kyphosis, as one observes with an emphysematous thorax.

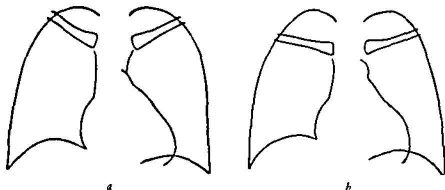


FIG. 51 —Mitral configuration of child's heart Thirteen and one-half year old boy (orthodiagram). The mitral configuration disappears with forward inclination of the upper part of the body (*a*) Upper part of body erect (*b*) Upper part of body bent forward

With descent of the diaphragm, the heart as a whole sinks caudad whereby the ventricular cone swings downward and mesial and simultaneously rotates around its long axis to the right Owing to this swing the heart becomes more oblique; its angle of inclination increases and in the erect position this amounts to 52 to 55

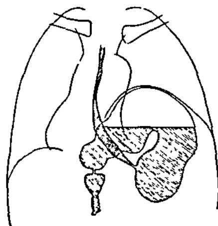


FIG. 52

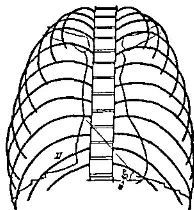


FIG 53

FIG 52 —Displacement of the heart to the right (dextroposition) from relaxation of diaphragm in a woman, 69 years old.

FIG. 53 —Diaphragmatic descent. The angle of inclination $\alpha > 45$ degrees. With extreme descent of the diaphragm, the eleventh rib can appear above the diaphragm. The diaphragm is flattened and frequently shows the notches of its costal attachments

degrees (Haudek) and in recumbent patients to 39 to 43 degrees (Dietlen). As with the transverse position, with an oblique heart the left border changes its position more than the right While the left border swings median decidedly and descends more sharply to the diaphragm, the right border, at most, shifts slightly inward, stretches, and elongates. Owing to this elongation the right ventricle can form the

diaphragmatic section of the right border (Assmann), a situation favored both by the obliquity as well as by rotation to the right. Sometimes a slight notch subdivides the right border into two arcs, the upper one corresponds to the right atrium and the lower to the right ventricle (Dietlen) (fig. 54).

Since both cardiac borders move inward, the transverse diameter Tr_H is diminished (fig. 39). Since the mid-left distance decreases much more than the mid-right, Ml approaches the value of Mr; this is called median placement. Such hearts are small, and, since the bulge of the left ventricular arc is reduced, have a shallow waist which creates an impression of a mitral configuration. This impression is enhanced by the fact that the aortic knob protrudes less to the left as the aortic loop stretches (p. 357). Often a flat-convex arc, bulging into the cardiac waist, completes its filling Schwarz interprets this arc as the left atrium rendered more prominent through

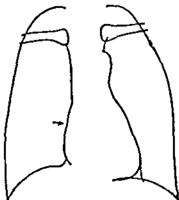


FIG 54.—Median placed heart with a flat protrusion of the conus pulmonalis on the left cardiac border, on the right side the right ventricle forms much of the cardiac border The notch (arrow) on the right cardiac border indicates the atrioventricular junction.

cardiac rotation to the right as mentioned above; Dietlen considers it the conus and pulmonary artery. Undoubtedly in many cases it actually represents the conus of the right ventricle (fig 54) and the pulmonary artery. The more distinct appearance and the length of the arc, often striking, certainly depend to some extent, upon the inward recession of the left ventricular arc and in part on the steeper course of the conus and pulmonary artery which otherwise seem very short owing to their dorsad course Whether these conditions should be regarded as a persistence of "infantile relations" (Bauer and Helm) or a vaguely defined cardiac inferiority (Kraus), as Dietlen assumes for higher grades of bulging of the left middle arc, must be left undecided.

With extreme descent of the diaphragm, the heart loses diaphragmatic support so that a space appears between it and the left diaphragm. These are called "dropped" (Kraus) or "pendulous hearts" (Wenckebach) (fig. 55).

There are, however, median placed as well as pendulous hearts even when the diaphragm is not low. It is assumed that such hearts, owing to their smallness or the abnormal brevity of the great vessels (fig. 264), especially of the aorta, stand so high that they rest imperfectly on the diaphragm (hypoplasia cordis et aortae, Kraus)

ance of the tenth or eleventh rib above the dome (fig. 53). The twelfth rib becomes visible only when the diaphragm is extremely flat and with coexisting kyphosis, as one observes with an emphysematous thorax.

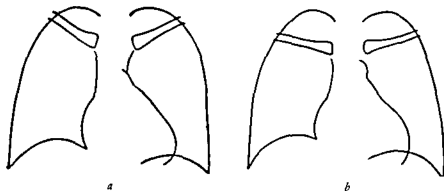


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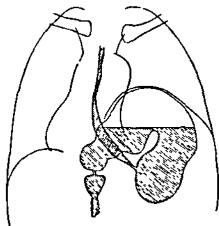


Fig 52

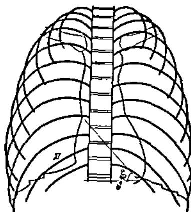


Fig 53

Fig 52.—Displacement of the heart to the right (dextroposition) from relaxation of diaphragm in a woman, 69 years old

Fig 53.—Diaphragmatic descent The angle of inclination $\alpha > 45$ degrees. With extreme descent of the diaphragm, the eleventh rib can appear above the diaphragm The diaphragm is flattened and frequently shows the notches of its costal attachments.

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exerted on the abdomen (Glénard's maneuver), when a firm binder was applied to the abdomen, or when the patient was examined in the recumbent position (Wenckebach, Kraus, Moritz, Dietlen, Bjure and Laurell). In the erect posture the volume of circulating blood in these patients was decidedly smaller than in the recumbent position and even fell to abnormal levels (Laurell). We have seen the circulating blood volume decrease 1.5 liters when these patients were studied in the erect position. In other words, in the upright position the heart receives abnormally small amounts of blood and consequently fills imperfectly. For this reason Wenckebach made improper support of the circulation by the low and flat diaphragm primarily responsible for the abnormally small inspiratory reserve, under these conditions only slight suction is exerted so that inadequate amounts of blood are moved toward the heart from the abdominal organs and from the liver in particular. If one adds to this, deficient thoracic respiration, commonly present with a flat, rigid, pear-shaped thorax, the return of blood to the heart definitely suffers (Wenckebach and Hofbauer). Besides these important mechanisms, diminished tonus of the splanchnic vessels (Kraus) and of skeletal muscles (Henderson) usually plays a decisive role Moreover, on standing, an abnormal quantity of blood remains in the abdominal vessels and in the dependent parts of the body. Consequently, orthostatic reduction of cardiac size is greater in vasolabile and asthenic individuals (Schiff, Dietlen), particularly in those with peripheral circulatory weakness of toxic or endocrine origin, even when the position of the diaphragm is normal. The thin and relaxed walls of these hearts favor their ability to diminish in size since adaptation to lower filling pressure proceeds with special ease by reduction of the chambers (Dietlen).

The vascular band of these hearts is also conspicuously small. Measurement of the aortic diameter usually yields abnormally low values so that one may wrongly assume the presence of aorta angusta. Repetition of the measurement in the recumbent position usually reveals, however, that the aorta also widens with enlargement of the heart and its dimensions may become normal, thus, the aorta is not actually narrow but like the heart is poorly filled when the patient stands (Zdansky) (fig. 45a)

The rate of this imperfectly filled heart is usually accelerated and on fluoroscopy its pulsations are lively. Especially with a pendulous heart, the apex shows large, lively systolic pulsations directed toward the base. The size of these pulsations is particularly striking since the stroke volume of these hearts is somewhat diminished Laurell explains this seeming contradiction as follows. The movement of the A-V septum, directed toward the apex in systole, is hindered and consequently the lateral walls and the apical sections of the ventricles make greater excursions against the atrioventricular septum from each side. This indicates how little the size of cardiac pulsations, visible roentgenologically, can be employed to evaluate cardiac stroke volume (Laurell)

Often the physical capacity of individuals with high grade orthostatic cardiac reduction is distinctly reduced Carriers of these hearts suffer from vertigo, momentary weakness, lassitude, headache, palpitation and a tendency to collapse (Wenckebach, Kraus, Dietlen, Laurell, Eppinger). Actually, however, they should not be considered "cardiacs" It is equally improper to designate these hearts as hypoplastic because they are relatively small and thin-walled as the result of the

Frequently the median placed heart of the ptotic individual is actually small. This smallness can be constitutional (true hypoplastic heart, Kraus) and usually corresponds to slenderness, poor musculature, and, frequently, anemia of its carrier (Dietlen). In a majority of cases, however, *the heart is normal in size but appears* small only because it is imperfectly filled in the erect position (Wenckebach, Kraus, Dietlen). (See further pages 103ff.)

Naturally there are no sharp limits between the oblique, transverse, and vertical heart. The same heart may pass from one to the other. A heart designated oblique in the final months of pregnancy may be vertical after delivery. To some extent one form may change to another by voluntary effort Haudek considers the oblique heart is a "normal situation" and recommends correcting the transverse or vertical positions, when possible, by inspiration or expiration to restore normality when analyzing the cardiac shadow Altstadt accepts this suggestion We shall return to this important and practical point in the discussion of the pathologic heart.

The significant alterations of the heart, in respect to the position and shape in the left and right lateral positions, from diaphragmatic changes and altered static conditions were mentioned on page 50 Reference should also be made to the chapter on adhesive pericarditis.

IX. The Small Heart

Not every heart which appears small on the screen is actually small A distinction must be made between the apparently small and the actually small heart At present we know that the hypoplastic heart, which is too small in relation to the body *carrying it, is relatively rare compared to the incidence of the apparently* small heart For this reason the latter will be discussed first

In the first place, abnormal smallness of the heart may be an illusion owing to its position in the chest. In the preceding section it was stated that diaphragmatic descent in ptotic individuals may reduce the cardiac shadow because the heart becomes median or vertical (p 101) Early, Dietlen noted that frequently, in these vertical hearts, the transverse diameter alone diminishes while precise measurements disclose that actual cardiac size remains entirely normal, even a pendulous heart by no means need be too small (Wenckebach) Experience also teaches that such individuals may be normal physically and can even meet great demands The small median heart is simply a partial expression of a definite body build, frequently characterized by a proportionate height, slenderness, low diaphragm, and often a flat chest. It is by no means the asthenic habitus of Stiller but may also be a racial type as exemplified by Frisians or Indians (Wenckebach). This failed to receive proper consideration for a long time and for this reason older reports on the incidence of *constitutionally* small hearts cannot be evaluated An exhaustive review of the relevant literature was published by Dietlen.

Naturally in many cases precise measurement of cardiac size actually showed the heart was abnormally small when studied in the erect patient. It would be premature, however, *to assume immediately and for this reason* that cardiac hypoplasia existed, for the cardiac shadow enlarged at once to normal size when pressure was

exerted on the abdomen (Glénard's maneuver), when a firm binder was applied to the abdomen, or when the patient was examined in the recumbent position (Wenckebach, Kraus, Moritz, Dietlen, Bjure and Laurell) In the erect posture the volume of circulating blood in these patients was decidedly smaller than in the recumbent position and even fell to abnormal levels (Laurell). We have seen the circulating blood volume decrease 1.5 liters when these patients were studied in the erect position In other words, in the upright position the heart receives abnormally small amounts of blood and consequently fills imperfectly. For this reason Wenckebach made improper support of the circulation by the low and flat diaphragm primarily responsible for the abnormally small inspiratory reserve, under these conditions only slight suction is exerted so that inadequate amounts of blood are moved toward the heart from the abdominal organs and from the liver in particular. If one adds to this, deficient thoracic respiration, commonly present with a flat, rigid, pear-shaped thorax, the return of blood to the heart definitely suffers (Wenckebach and Hofbauer) Besides these important mechanisms, diminished tonus of the splanchnic vessels (Kraus) and of skeletal muscles (Henderson) usually plays a decisive role. Moreover, on standing, an abnormal quantity of blood remains in the abdominal vessels and in the dependent parts of the body Consequently, orthostatic reduction of cardiac size is greater in vasolabile and asthenic individuals (Schiff, Dietlen), particularly in those with peripheral circulatory weakness of toxic or endocrine origin, even when the position of the diaphragm is normal. The thin and relaxed walls of these hearts favor their ability to diminish in size since adaptation to lower filling pressure proceeds with special ease by reduction of the chambers (Dietlen).

The vascular band of these hearts is also conspicuously small. Measurement of the aortic diameter usually yields abnormally low values so that one may wrongly assume the presence of aorta angusta Repetition of the measurement in the recumbent position usually reveals, however, that the aorta also widens with enlargement of the heart and its dimensions may become normal, thus, the aorta is not actually narrow but like the heart is poorly filled when the patient stands (Zdansky) (fig. 45a).

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diminished demands usually placed upon them by individuals poorly equipped for physical activity and with weak muscles. Subsequently, these hearts may regain normal size and shape and lose their marked orthostatic reduction when physical training improves the state and tonus of skeletal muscle, raises the diaphragm, and improves peripheral vascular tonus. This increase of size depends primarily upon better cardiac filling and only secondarily upon an increase of muscle mass.

One should consider the heart small, hypoplastic, and inferior by virtue of its anlage only when it is small in both the erect and recumbent positions, that is, despite optimal conditions for filling, the smallness should not depend upon peripheral vasomotor weakness or reduction of blood volume from marked dehydration



Fig. 55 —Hypoplastic heart Cor pendulum

(diarrhea, cachexia) Hearts, too small by virtue of their anlage, are much rarer (Wenckebach, Dietlen) than was formerly assumed. Often they appear as cor pendulum or a dropped heart (fig 55) Usually they are a single feature of the degenerative habitus (Kraus) Moreover, they are by no means always median or vertical since the thorax of these individuals is often not elongated, on the contrary, it is too short and the central tendon is relatively high, the diaphragm simply descends less abruptly on all sides These hearts lie abnormally far from the anterior chest wall so that with frontal passage of ray, the retrosternal field is broad (Kraus). Often the vascular band is conspicuously short. Measurement of the aortic diameter in the recumbent position usually gives low values (1.8 to 2.3 cm for the arch of adults measured by Kreuzfuchs' method) with a true hypoplastic heart so that a hypoplastic aorta (aorta angusta) must be assumed (Zdansky).

A true hypoplastic heart alters its size and shape very little during the course of life unless it suffers from dilatation (over-exertion, thyrotoxicosis, anemia, infection). At most, the hypoplastic heart may become "globular" (Kraus), this happens mainly at more advanced ages, partly owing to left-sided hypertrophy, partly from tilting into a transverse position by the atheromatous, elongated aorta. In the roentgenogram the small cardiac shadow bulges hemispherically to the left below a narrow, often elongated vascular band, the angle of inclination can be greatly reduced owing to the transverse position (fig. 56).

Kraus thought that the hypoplastic heart might be too small only in respect to a certain dimension of the body and later it could attain normal size This assumption seems to efface the limits between a hypoplastic heart as determined by its anlage, and the heart which is too small by virtue of poor development and lesser use of

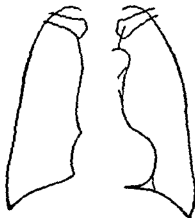


Fig 56

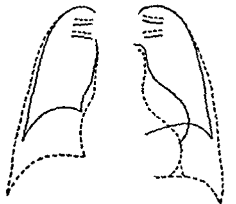


Fig 57

Fig. 56.—"Globular heart" in an old man with atheromatous elongation of the aorta

Fig 57.—Alterations of the cardiovascular shadow from deep breathing (_____deep expiration, - - - - -deep inspiration)

skeletal muscles, in the latter the heart merely appears small owing to inadequate filling. If, by physical training, a small heart grows to normal size, one can scarcely speak of constitutional hypoplasia. At most this involves an individual in the process of growing, whose heart, for constitutional reasons, lagged behind general growth and subsequently attained proper size. The adult hypoplastic heart, on the contrary, should enlarge only from dilatation resulting from myocardial failure and not by harmonious growth of its chambers and walls.

Some writers declare that no sharp boundary separates the small heart of ptotic-asthenics from the hypoplastic heart of Kraus (Bauer). Certainly a definite decision in single cases is not always possible. Nevertheless, one may assume with some probability that a constitutional hypoplasia of the heart or aorta is present when the cardiac or aortic shadow, respectively, remain small in the horizontal position. If, on the other hand, the small heart and the aorta of a ptotic individual enlarge to normal in the recumbent position, if the heart attains normal size with physical training and then shows less orthostatic reduction than before (Moritz), in our opinion, a constitutional hypoplastic heart is not present.

It is clear that a constitutionally small heart is by far less common than earlier authors assumed. Rather, it usually involves a situation in *which static factors are* able to act upon the heart to a greater degree in the sense of orthostatic reduction of cardiac inflow by virtue of special peripheral conditions.

Dynamic forces may also play a decisive role in reducing the return of blood to the heart. This happens, for example, with defective respiration resulting from flattening of the diaphragm. In monkeys, suitably fastened to a centrifuge, centrifugal force acting in a craniocaudal direction, such as might be effective in aviation, can reduce cardiac size and narrow the great vessels as the result of diminished influx of blood (Fischer and Pfeiffer).

X. The Heart and Respiration

In respiration, the heart experiences alterations of position, shape, and size which can be most thoroughly observed roentgenologically. These depend partly upon spatial intrathoracic changes and partly on differences in cardiac filling.

Since the heart is fixed by the pericardium to the diaphragm and rests on it, the heart must definitely change position with respiratory shifting of the diaphragm. *These changes are rather insignificant when respiration is quiet. With deep breathing*, however, through inspiratory descent of the diaphragm the heart becomes vertical and with expiratory elevation of the diaphragm, it is displaced upward and assumes a transverse position (fig. 57). Accordingly, the heart as a whole simultaneously moves caudad or craniad, respectively, and, furthermore, the parts of the heart near the diaphragm shift more than those located elsewhere (Moritz). Since the respiratory excursions of the diaphragmatic domes are essentially wider than those of the central tendon on which the heart rests, as the domes ascend in expiration the heart sinks more deeply into abdominal shadow, inspiration lifts the heart out of this shadow so that the apex may appear above the diaphragm. Respiratory shift affects the great vessels least. If the distance between the aortic knob and the clavicles increases in inspiration, descent of the aortic arch is less responsible than *inspiratory elevation of the shoulder girdle* (Frik).

Apart from these positional alterations resulting from altered spatial conditions, the cardiovascular shadow undergoes various changes in shape. These depend in degree not only upon the extent of diaphragmatic respiratory shift but differ qualitatively according to the resistance the heart opposes to transformation. If the cardiac walls are relatively thin, the vessels delicate, and the mediastinal connective tissue, as well as pleura, yielding, deformation of the mediastinum by compression dominates. In expiration, the heart is compressed to some extent in a craniocaudal direction. Both of its borders, the left more than the right, bend outward and become rounder, the cardiac waist becomes shallow or may even vanish so that the cardiac shadow appears less differentiated, it may even approximate a mitral configuration. The same picture as that *often found in young women and children with relative* diaphragmatic elevation can develop (fig. 50b). If the heart muscle is powerful and thick, the vessels thick or rigid, and the mediastinal connective tissue and pleura firm, deforma-

tion of the cardiovascular complex by rotation and transverse position dominates over compression (fig. 50a). Hearts which are not easily compressed by expiratory shortening of the thorax must yield, in some other way to pressure exerted from below by the diaphragm. Since cephalic movement of the entire heart is possible only to a limited extent, a shift can take place only through rotation This rotation is essentially a swing of the longitudinal cardiac diameter around a fulcrum located near the right cardiovascular angle which also moves slightly upward. Simultaneously, both limbs of the aortic arch, the ascending and descending portions, are pushed apart so that the vascular band broadens and the aortic knob protrudes farther to the left. The resultant changes are the same as those otherwise occurring with diaphragmatic elevation, namely, transverse position of the heart, a smaller angle of inclination, predominant increase of the mid-left distance, greater rounding of the right cardiac border, widening of the vascular band, increased prominence of the aortic knob, and deepening of the cardiac waist, in short, a more or less distinct aortic configuration.

In inspiration the cardiovascular shadow stretches so that the mid-left distance decreases decidedly, the mid-right to a lesser extent If the cardiac shadow had a mitral configuration owing to compression during expiration, a distinct cardiac waist now appears, if a transverse position has given the cardiac shadow an aortic configuration, the cardiac waist becomes shallow.

As mentioned earlier, these changes are minimal or absent with quiet respiration They can be disregarded for practical purposes (Groedel and Moritz) With deep breathing, however, they are very conspicuous Dietlen correctly emphasizes that in the erect posture the expiratory changes in cardiac position and shape are more evident than the inspiratory since in this posture the diaphragm is located nearer the inspiratory position from the start On the other hand, when patients are recumbent, the opposite situation prevails. Similar conditions exist with diaphragmatic descent or elevation, respectively. In the first case the expiratory reserve is greater and expiration affects shape and position most, in the second case the inspiratory reserve is greater (Haudek) and changes occur mainly in inspiration

The changes of cardiac volume induced by respiration merit special mention Practically, they come under consideration only in deep breathing On the basis of cinematographic observations, Groedel calls them trifling, in his opinion they may be merely illusory in a majority of cases, owing to changes of cardiac shape and position. Oestreich and de la Camp, as well as Moritz, believe that reduction of cardiac size is common with deep inspiration owing to pericardial tension and easier emptying of the right heart into the lungs Zuntz and Schumberg, Holzknecht and Hofbauer, Wenckebach, Eppinger, and Arkussky, on the contrary, observed cardiac volume increase in inspiration and they ascribed this to augmented cardiac filling at this time. Subsequently, Moritz noted inspiratory enlargement of the heart in bronchial asthma; he placed strong emphasis upon the abnormally high intrathoracic pressure, the result of impeded expiration, which, like a Valsalva test (see below) should produce an expiratory reduction of cardiac volume.

More recent investigations indicate that inspiratory reduction of cardiac volume scarcely comes under consideration The reduction of heart size tends to be only an illusion since the heart loses width and stretches from diaphragmatic descent

(Groedel) Certainly cardiac acceleration often accompanying inspiration is not sufficient to reduce heart volume distinctly.

Theoretically the opposite situation should prevail in inspiration, namely, cardiac enlargement, more blood flows into the heart at this time. This enlargement is offset, to be sure, for the left heart obtains less blood as more is retained in the lungs owing to decreased intrathoracic pressure. On fluoroscopy often the superior vena cava and the right border of the heart actually expand with each deep inspiration as the result of an augmented influx of blood, while the left heart, owing to diminished return of blood from the lungs and its vertical position in inspiration, moves median. In these cases both cardiac borders move to the right in inspiration and return to the left with succeeding expiration. In other words, the cardiac shadow may show a respiratory pendulum movement (Laurell) as roentgenkymograms suggest (Zdansky

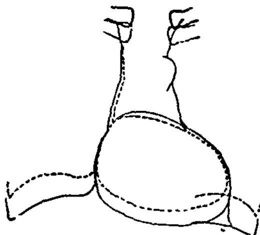


FIG 58 —Increased cardiac size in inspiration in a case of severe emphysema with cardiac decompensation. (———inspiration, ----- expiration)

and Ellinger, Weltz). Often this movement is particularly distinct when the patient sniffs (Zdansky) This "pseudomediastinal wandering" should not be confused with true wandering which may occur, for example, in bronchial stenosis. The barium filled esophagus does not participate in the movement (Zdansky).

Definite inspiratory cardiac enlargement is found only with stenotic breathing, with brisk, forced inspiration in patients with emphysema (fig 58) and particularly in an attack of bronchial asthma (Moritz). Under these circumstances an unequivocal expansion of both cardiac borders can suggest real cardiac distension Roentgenkymographic analysis (Zdansky and Ellinger) reveals a sudden enlargement of both halves of the heart in inspiration which gives way to an equally sudden and simultaneous reduction in expiration The synchronous alterations of left and right heart volumes strongly implies that changes of intrathoracic pressure have outstanding causal significance Weltz confirmed this.

Most impressive among respiratory alterations of cardiac volume are those resulting from suction and pressure (Muller and Valsalva tests). They have been known for a long time (v. Criegern, Kraus, Moritz, Dietlen, de la Camp, Natvig, Nordenfelt) and were analyzed by roentgenkymography (Zdansky and Ellinger,

Fetzer, Nolte, Klaus and Albert, Teschendorf, Moberg, Bordet and Fischgold, and others) so that their course now is clear.

In Muller's test after deep expiration the mouth and nose are closed and a maximal inspiratory effort is attempted to produce a considerable reduction of intrathoracic pressure. Simultaneously, with sudden lifting of the ribs and jerky descent of the diaphragm, both cardiac borders jut out and become more rounded. The outward thrust, usually more on the right than the left (Fetzer), is synchronous on both sides with the beat immediately after suction begins Thereby, the diastolic inward movement of the right cardiac border, indicating the atrium emptying into the ventricle (p. 59), may vanish or may be replaced by a wider lateral movement as blood shoots into the atrium (Zdansky and Ellinger). The left ventricular arc which also moves laterad with the first diastole retains this augmented mid-left distance with reduced pulsations during the entire period of suction. Roentgenographically, Fetzer recorded a retrograde systolic atrial wave on the superior vena caval shadow and reduced pulsation of the aortic knob Furthermore, lung vessel markings are intensified as the blood content increases, the latter was confirmed by Pfeiffer. The shadow of the azygos vein in the right trachobronchial angle may enlarge (Lutz).

Undoubtedly the heart expands at the beginning of suction owing to the sudden influx of blood Since this expansion persists throughout suction, the heart must retain more residual blood under the influence of lower intrathoracic pressure This idea finds support in the observation that immediately after suction ends, both cardiac borders synchronously move median to restore the original volume of the heart after 1 to 2 beats

In the Valsalva test, intrathoracic pressure is increased by the subject exerting pressure after deep inspiration. When pressure begins the cardiac shadow diminishes on all sides, as a rule, with a simultaneous increase of rate. Finally, the heart can become so small that it seems to pump empty (Kraus). In these cases blood pressure may decline too low for measurement, with signs of acute cerebral anemia and a pulseless radial artery. This happens predominantly in ptotic-asthenic individuals; Burger calls them syncopotropic. In one patient Narvig calculated that the cardiac volume was reduced about 390 cc. A transient cardiac enlargement may precede the reduction, perhaps, a result of simultaneous expulsion of blood from the large veins in front of the ventricles into the right and left heart (Nolte). Usually the reduction of heart size is striking, this increases from one beat to the next (Bürger) and affects both halves of the heart simultaneously. Undoubtedly the impeded influx of blood into the heart and heightened intrathoracic pressure places a load on the heart and causes it to beat empty (Nolte). As long as blood content is decreased, the hilar shadows become smaller and markings in the lung fields become small, short, and sparse (Pfeiffer).

While the cardiac shadow is reduced in size and visible pulsations become noticeably smaller, they do not disappear (Zdansky, Kraus, Nolte). Stumpf observed disappearance only in exceptional cases. The alterations of pulsations were studied exactly with roentgenkymography (Klaus and Albert) (p. 112).

When pressure ends, both borders of the cardiac shadow immediately move laterad and the original cardiac size is restored after two or three beats (de la Camp,

Zdansky and Ellinger, Nolte). Once Zdansky and Ellinger recorded pulsations of an alternans type on the left ventricular arc in the post-pressure period. Occasionally Nolte observed very large, slow pulsations during the post pressure bradycardia (Burger)

The extent of cardiac enlargement in the Muller test and of cardiac reduction in the Valsalva experiment varies greatly with different individuals. Occasionally the cardiac shadow even enlarges (paradoxic Valsalva effect) during the entire period of pressure (v. Criegern, Klaus and Albert) (p 112).

Under the assumption that cardiac volume changed with equal increases or decreases of intrathoracic pressure, manometrically measured, and that these depended upon the cardiac status, attempts were made to utilize suction and particularly forced respiration as a functional test of the heart (p. 129), to obtain insight into the state of the myocardium and particularly its tonus.

XI. Is Any Tonus-Function of the Heart Roentgenologically Demonstrable?

Alterations in cardiac size and shape in the Muller and Valsalva tests lead directly to a discussion of the question just posed

Readers, roentgenologically oriented, hearing of cardiac tonus, think particularly of the work of Zehbe, Pongs, Plaut, and Dietlen. Zehbe introduced the conception of cardiac tonus into roentgenology by relating it to varying consistency of the myocardium at postmortem, this concept is tenable physiologically. By means of the roentgen image he distinguished normal, relaxed, and hypertonic hearts according to the extent that shape and the alteration of the angle of inclination changed with deep expiration. The relaxed (hypotonic) heart widened with deep inspiration into a shapeless mass like a "lump of dough" over the elevated diaphragm while its angle of inclination became progressively smaller, normotonic hearts showed much less change while hypertonic hearts showed no change in shape or in the angle of inclination and created the impression that the heart bored into the diaphragm.

Pongs and Plaut confirmed the essentials of these observations and concurred in Zehbe's interpretation. Pongs distinguished a "relaxed" and "taut" heart by a "plate" test, as he called it, and by the Valsalva experiment He quotes Zehbe almost verbatim when he states that the "relaxed" heart in deep expiration spreads on the elevated diaphragm (the plate) like "dough." Another sign of the "relaxed" heart is a marked reduction in size in the Valsalva test The "taut" heart, on the contrary, preserves its shape in deep expiration and shows no noteworthy reduction in size with pressure.

Dietlen was deeply occupied with the tonus problem and made an extensive report on this question. He concurred with the writers just named in regard to cardiac tonus as a property of the myocardium "which determines its shape in the state of relaxation and its stabile form despite opposing influences " He also considers it reasonable to refer increased lability of cardiac size and shape to reduced diastolic tension of the myocardium Since hypertrophied hearts generally preserve a more constant form than normal or dilated ones, he attributes hypertonic properties

to hypertrophied hearts. Dietlen justifies this conclusion by the probability that, *a priori*, a strong hypertrophic myocardium embraces its content more strongly during diastolic relaxation than normal or dilated muscle. Nevertheless, Dietlen expressly states that not every dilated heart is relaxed and hypotonic and that cardiac relaxation is not necessarily combined with dilatation. Rather, roentgenologic signs of relaxation are absent in many dilated hearts and it is precisely the small hearts which are seemingly relaxed. The types of cardiac dilatation showing signs of relaxation are left undecided although Dietlen believes that dilatations which are the result of acute infections and toxic injuries are primarily responsible.

Obviously views concerning demonstrability of cardiac tonus by x-ray are based on the observation that stability of cardiac shape and size quantitatively is subject to marked differences and on the idea that preservation of size and shape is determined by the status of myocardial tension in diastole (tonus). These premises seemed to compel the following conclusion a heart labile in respect to size and shape possesses less tonus and less efficiency than one which opposes greater resistance to such changes. Accordingly, Plaut felt justified, merely from the positive outcome of his roentgenologic test of tonus, to conclude that the myocardium is damaged or at least inferior even in the absence of clinical signs of such damage, Dietlen also inclined to the view that a heart, relaxed on the basis of these tests, should be considered functionally inferior.

The conception of lessened efficiency in association with reduced stability of size and shape also seems to find support in connection with hearts dilated by acute infections and toxins (Dietlen) as well as in those whose carriers, in accordance with their ptotic-asthenic constitution, are less efficient, these hearts are prone to display lability of size and shape

Despite the truth of the roentgenologic observations cited, many facts and considerations make it dubious whether lability of cardiac shape and size can be considered convincing proof or even an indication of diminished tonus and efficiency of the myocardium First among the relaxed hearts are the "small" ones (Dietlen). These "small hearts" are encountered most often in ptotic-asthenic individuals who are poorly equipped for physical exertion and are less efficient, but usually, for reasons shown on page 102, the heart is not actually small but only seems so for extracardiac reasons (defective diaphragmatic function, peripheral vasomotor weakness, reduced tonus of the skeletal musculature) and, indeed, only when improperly filled with blood When a heart, subjected to decreased filling pressure, becomes particularly small in a Valsalva test and pumps almost empty, when it spreads broadly on the elevated diaphragm in Pong's plate test, relaxation of the myocardium should not be made responsible without further consideration Rather, lability of cardiac size and shape can be explained with less compulsion by inadequate filling which makes the heart more susceptible to changing intrathoracic spatial and pressure conditions than when normal filling pressure supplies it with normal volumes of blood (Zdansky) The same holds for the lability of cardiac size and shape in infectious diseases and toxic states, in endocrine disturbances and some central and peripheral diseases of the nervous system.

Apart from the factors already mentioned and involving the peripheral circulation, lability of cardiac size and shape can be accentuated in many of these patients

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the systolic pressure and volume efficiency, is inadequate to explain this quantitatively. It seems to him that adaptation of volume is associated with much greater difference of presystolic length of fibers than is the mechanism of tension adaptation, in other words, volume efficiency is associated with greater variations of presystolic ventricular volume than one should expect according to the Starling-Frank laws

In this conception, decreased tonus does not unconditionally express diminished myocardial efficiency for it may also be compensatory if the heart is to exert a greater stroke by means of greater relaxation and tension.

The property Hess designates as tonus is the same, at least in part, as the "contraction debt" of Straub, it is conceptually different from actual tonus but fails to separate sharply these two forces from each other and their effects on the active heart.

Certainly myocardial tonus can appear only during diastolic relaxation in that it affects the degree of diastolic filling and the course of diastolic movement. Perhaps, this provides the possibility of a roentgenologic approach to the problem of myocardial tonus Reduced tonus would permit greater diastolic widening, that is, would orient the heart to large diastolic volumes, the return to normal tonus or to an abnormal increase of tonus would act in the opposite direction. Actually some alterations of cardiac size have been connected with alterations of tonus, thus, the larger heart volume after exertion has been ascribed to a compensatory or even decompensatory reduction of tonus (Rautmann, Walko) and reduction of cardiac size after work to heightened tonus (Ewig, Schenk, Rautmann) In these volumetric alterations a variety of other factors (volume of circulating blood, filling pressure, pulse rate) drastically influence the extent of diastolic filling. Only those changes in cardiac volume should be interpreted as alterations of myocardial tonus which are observed under otherwise identical conditions (constant blood pressure, equal volumes of circulating blood, minute volumes and pulse rates). Apparently such observations have not been conducted systematically.

Perchance, the effect of myocardial tonus can be inferred from changes in the visible ventricular pulsations and those recorded by kymograms. One might think that reduced diastolic tension might make outward diastolic movements of the ventricular wall more brisk and perhaps also might lead to the appearance of an abnormal sequence of movements in the diastolic pulsation period. The strikingly lively, almost flapping cardiac excursions observed in acute and chronic infectious diseases, especially in endomyocarditis, in severe primary and secondary anemias, after physical exertion, mental excitement, in neuroses, hyperthyroidism and after warm baths, make it likely that this "excited cardiac action" could depend upon reduced tonus, but peripheral vasomotor weakness or diminished return of blood to the heart (Laurell), hyperthyroidism, mental excitement, work, or warm baths also accelerate the circulation greatly and increase stroke volume. They may produce large and lively pulsations along the cardiac borders. If it is true that enlargement of the stroke volume is induced by reducing myocardial tonus (Hess, Straub, Rautmann), the "excited action" of these cases would be closely connected with myocardial tonus

We endeavored to ascertain by means of roentgenkymograms what alterations of ventricular pulsation might be referred to changes of diastolic myocardial tension. We

by the status of the heart itself, particularly when its walls are thin. If myocardial thinness were equivalent to low tonus, as Dietlen asserts, this would mean that the lability of size and shape of the small hearts of asthenic individuals actually is conditioned, at least in part, by reduced cardiac tonus. Nevertheless, it is doubtful whether decreased tonus in this sense can be interpreted as myocardial inferiority without further qualification because the thinness of the wall may merely reflect the slight demands these individuals impose upon their hearts

This is not intended to deny that lability of cardiac size and shape in many toxic-infectious states as well as in circulatory disturbances of endocrine and nervous origin cannot contribute to reduced diastolic myocardial tension This seems highly probable since the heart, as part of the vascular system (Rothberger), should participate in a hypotonic orientation of the entire vascular system

Albert and Klaus believe that conclusions regarding myocardial tonus can be drawn from alterations of cardiac size (deduced from roentgenologic cardiac area) by a manometrically measured increase of intrathoracic pressure amounting to 20 to 60 mm Hg. They set up four types of reaction

Type I Relatively marked reduction of cardiac size with an increase of pressure (20 mm Hg) and only slight further reduction with 40 mm. Hg is found in relatively large hearts of young athletes, as a rule, it speaks for "moderate to average myocardial tonus but sometimes also occurs with a large heart and in Burger's "syncopotropic type "

Type II Relatively slight reduction of cardiac size with a 20 mm Hg increase of pressure but marked additional reduction with 60 mm Hg is found with average and above average hearts and favor "good myocardial tonus or hypertrophy" respectively and excludes dilatation

Type III No further cardiac reduction when pressure increases from 20 to 60 mm Hg, this intermediate between type I and II is found with relatively large hearts of well trained athletes In them it represents a "favorable type of reaction "

Type IV A slight reduction with a pressure of 20 mm Hg and an enlargement when pressure is increased to 60 mm Hg is ascribed to copious influx of blood from the abdominal cavity by augmented intra-abdominal pressure

The conclusive nature of these reaction types for varying myocardial tonus is highly problematic owing to the interplay of peripheral regulations and the incalculable influence of abdominal pressure activated by the Valsalva test. On the basis of Nordenfelt's studies, Moberg correctly notes that the effect of abdominal pressure may be equal to or greater than intrathoracic pressure so that the cardiac volume reducing effect of the latter is abolished or even over-compensated (paradoxic Valsalva effect) In other words, absolute certainty in respect to the cardiac tonus cannot be gained from the Valsalva test (Frik and Natvig).

Endeavors to infer cardiac tonus by roentgenologic methods also suffer from the obscurity surrounding the nature of myocardial tonus. Some consider the very

filling. Tonus determines the nadir reached by diastolic relaxation and the behavior of tension during the entire diastolic phase as well as the velocity with which the myocardium again relaxes after the expulsion period (Hess).

Hess maintains it is necessary to assume tonus. In his opinion, Starling's "law of the heart" which expresses the simple relation between presystolic tension and

Zuntz observed that the cardiac enlargement during work receded within three seconds

De la Camp, L. Raab, de Agostino, Rautmann, and Williamson labelled enlargement actually a sign of an abnormal or, at least, inferior heart. These statements naturally are in disagreement with other findings (Nicolai and Zuntz, Bruns and Römer) which suggest that the reduction of cardiac size is not the sole nor regular result of a single physical effort. In teleroentgenograms taken two to five minutes after performing exercise (Bruns and Romer), 75 per cent showed a reduction of heart size, but 7 per cent an enlargement and 18 per cent an oscillation between enlargement and reduction. Rautmann and Duras also observed such fluctuations. Soon several similar reports appeared. In twenty-six marathon runners Ackermann found enlargement in six but in the others a more or less marked reduction of cardiac size, if one reads the protocols of de la Camp, in which a reduction is considered merely a normal reaction, among the individuals regarded as normal from a cardiac and circulatory standpoint, increases of cardiac breadth occurred which greatly exceed the limits of orthodiagraphic error. McCrea, Eyster, and Meek also found regular reduction of cardiac size only from moderate effort and brief heavy work, after long, hard work on a cycle ergometer, the enlargement might persist for five minutes.

Owing to the prevailing uncertainty and seeming contradictions, Zdansky studied roentgenologically changes of cardiac size and shape after single efforts (treading a cycle ergometer or climbing stairs) by untrained subjects, the alterations of cardiac size were followed for a long time by calculating cardiac volume by the Rohrer technic The following results were obtained

1. Increased cardiac size immediately after the exercise which, under some conditions, lasted for several minutes,

2 Decreased cardiac size immediately after work or after an initial enlargement, identical with the postexertional reduction demonstrated by Kienbock, Selig and Beck, and others,

3. Fluctuations of cardiac volume usually connected to the phase of reduction but occasionally occurring immediately after exercise and exceeding the original volume of the heart

These alterations of cardiac volume were not identical in all individuals, moreover, their extent, duration, and even appearance depended upon certain conditions

The initial increase of size consists mainly in cardiac elongation Since increased peripheral resistance scarcely comes into consideration in this phase of effort, the larger size of the heart must depend upon augmented influx of blood lasting beyond the period of exercise Obviously, in many cases this influx may oppose the effects of acceleration and increased contraction which tend to reduce cardiac size so that even cardiac enlargement may occur. This assumption is supported by the fact that the initial increase of size is particularly common when the study is undertaken in an individual who at rest shows a marked orthostatic reduction of cardiac volume In these cases the cardiac enlargement rather regularly persists for minutes above the resting value obviously because the heart receives more blood owing to deeper respiration and increased tonus of peripheral vessels and skeletal muscles than it does at rest. Hearts without noteworthy orthostatic reduction are well filled even

Finally, the thin-walled heart of the untrained person, as the result of greater distensibility and lesser power of contraction, may dilate relatively more from the augmented influx of blood and increased resistence than the thick heart of a trained subject (Bainbridge), this indicates how complicated the situation is and shows that no absolutely constant change of cardiac size during work should even be expected

The absence of a constant relation between changes of cardiac size on the one hand and of cardiac rate and blood pressure on the other depends (Bruns and Romer) upon reflex and hormonal influences originating in the central nervous system Naturally emotional influences cannot be neglected.

Moreover, the kind of physical exertion certainly influences alterations of cardiac size (Rautmann) Short-lasting efforts performed after deep inspiration with breath holding under conditions like the Valsalva test can reduce cardiac size unless this is offset by a simultaneous rise of intra-abdominal pressure by powerful activation of abdominal muscles as well as an acute rise of peripheral resistance Long-lasting performances like long distance running, swimming, and rowing, which are accompanied by uniform deep breathing, utilization of large groups of muscles, and massive influx of blood into the heart, by emptying blood depots and short-circuiting inactive parts can be expected to dilate both halves of the heart and especially the right side with its weaker muscle

Reliable reports on alterations of cardiac shape during work are not available.

As we have already mentioned, pulsations of the cardiovascular shadow during work not only become more frequent but they exhibit "excited action" (Dietlen) This results from increased diastolic influx of blood into the heart and augmented systolic ventricular contraction (Krehl) and is an expression of greater stroke volume It is the correlate of the increased apical impulse and widespread shaking of the chest wall (Moritz) It lasts somewhat longer than the exercise and ends approximately with the tachycardia. Surface roentgenkymograms (Stumpf) reveal that the breadth of excursions increases on all sides, indeed, the places along the cardiac border with small excursions during rest display relatively larger increases than areas subject to greater movement at rest The diastolic limb of the curve in the ventricular kymogram is relatively more vertical than the systolic, this corresponds to *the well known fact that tachycardia takes place mainly through shortening* of diastole Finally, atrial contractions, in contrast to the ventricular, recede more and more

2. *Alterations of the Roentgenogram after Single Physical Performances*

The first roentgenologic studies on the effects of this kind of exertion were conducted by Schott who thought cardiac size increased. This observation was partly confirmed by Lipschitz but was soon vigorously denied by Moritz, de la Camp, and others, their objections were raised primarily against Schott's method of study and actually the figures reported by Schott do not support his conclusions. Later investigators (de la Camp, Moritz, Dietlen, Kienbock, Selig and Beck, L. Raab, de Agostino, Williamson, Rautmann, McCrea, Eyster and Meek, R. and E Paterson, among others) found reduction of cardiac size a normal reaction. Gordon and Strong arrived at the same conclusion *in animal experiments. Nicolai and*

exercise by hours, days, and even weeks (fig 59) (Dietlen, Bruns and Römer, Zdansky) and may recur each time exercise is undertaken. Usually these enlargements are not permanent but tend to recede sooner or later (Dietlen).

Cardiac enlargement after exercise has utmost practical interest Most authors interpret it as a sign of impaired cardiac strength (de la Camp, Dietlen, Moritz, L. Raab, Williamson, Bruns and Romer, Rautmann, and others) De la Camp actually found such enlargement more common and greater in vasolabile individuals and those who had passed through an infectious disease, in fever, anemia, diabetes, and valvular diseases Rautmann saw cardiac volume increase acutely in a runner who collapsed after a race. Williamson also found enlargement which he interpreted as a sign of decreased cardiac strength We have noted trifling increases in patients with coronary sclerosis and exertional angina pectoris after bending the knees or climbing stairs Analogous observations (Reindell) imply that enlargements outlasting the exercise may be produced by diminished cardiac strength.

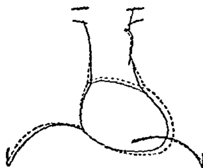


FIG 59—Increase of vertical heart size after swimming practice ——before practice cardiac volume, 452 cc (Rohrer method), ------twelve hours after practice cardiac volume, 508 cc (Rohrer method). Youth, 16 years old Height 174 cm, weight 76 Kg

On the other hand, Bruns and Romer emphasized that hearts enlarging after an exercise test, *subsequently showed normal efficiency and that if exercise is systematically continued*, the work can, secondarily, cause hypertrophy They found it more frequently in young asthenic individuals and in the so-called "illy-developed giant," in "constitutional cardiac weakness," and in individuals with median placed or with pendulous hearts, in weak, anemic, nervous people From similar observations Zdansky *gained the impression that young individuals with marked orthostatic reduction of cardiac size at rest were the ones whose hearts particularly enlarged after exertion Often this enlargement involved exclusively or predominantly* the volume in the upright position while the volume in the recumbent position was affected less or not at all (fig 60a and b) In no instance could one speak of a cardiac enlargement beyond normal, rather, with exercise the heart attained, and maintained for a while, a size suitable for the carrier Actually, in these cases only abnormal orthostatic reduction was abolished and it is at least highly probable that peripheral influences play a role in the sense that by greater vasomotor and skeletal muscle tonus, blood is better distributed through the body and the influx into the heart increases.

at rest and usually increased volume is not observed two minutes after the exercise; this is comprehensible when one considers how rapidly the augmented circulating *blood* and cardiac stroke volumes return to resting values after exercise (Zdansky)

At all events, reduction of cardiac size after exercise is not merely the result of tachycardia diminishing cardiac volume, it can persist long after the rate subsides to its original value and is even observed *without noteworthy acceleration (Bruns and* Romer). Likewise no relation is found to changes of blood pressure

Even simple fluoroscopy suggests that pulsations of the cardiac shadow not only are more frequent and livelier but are also larger than at rest Actually roentgenkymography shows that in normal hearts the breadth of ventricular pulsations ("movement space," Stumpf) have grown and this suggests increased stroke volume Moreover, the systolic as well as diastolic diameters of the heart become smaller, the first more than the second (Stumpf, v. Braunbehrens, and Reindell) The systolic reduction may result from more complete systolic emptying consequent to stronger contractions (increased accelerans tonus), the diastolic reduction, on *the whole, may depend mainly upon shortening of the filling phase and possibly on a* reduced capacity of the heart for diastolic dilatation, the consequence of augmented "contraction debt" (Straub, Rautmann)

Not only forces active in the heart itself but also peripheral factors may be responsible for the reduction in cardiac size Kienbock, Selig, and Beck suggest "dilatation of certain pathways" Straub also considered a relaxation occurring particularly in the splanchnic vessels. Finally, the extent of reduction of cardiac size and even its appearance is profoundly influenced by the amount of blood returning from the periphery as well as cardiac filling at rest (Zdansky). The postexertional period with its diminution of cardiac size may summate with one ortho*statically conditioned and may provoke greater reduction in the standing* than in the recumbent patient On the other hand, hearts inadequately filled even in the resting period would escape further reduction if this were equivalent to complete cardiac emptiness Naturally this event would be prevented by appropriate counterregulations

Reduction of cardiac size may last for minutes, hours, or even days after a single strenuous exertion. After a brief burst of speed (a thousand meter race), there was rapid subsidence while after a longer performance *with relatively less work per* unit time (long distance running), the reduction of cardiac size persisted longer (Rautmann). Factors in the myocardium or acting directly on it, perhaps, may provoke the changes in shape noted by Zdansky, they may be associated with changes of volume but they also occur without them

The increase of cardiac volume following reduction usually restores the original volume and may exceed it Often repeated fluctuations between enlargement and renewed reduction occur (Rautmann and Duras, Zdansky) *To what extent over*shooting of increased volume and fluctuations of cardiac size depend upon alterations

Cer-
least
reflex

changes in the flow of blood into the heart (Zdansky).

Likewise cardiac enlargement exceeding the resting volume may outlast the

largement since better capillarization and arrangements for oxidation in the skeletal musculature counteracted the increased cardiac work. Since cardiac hypertrophy and dilatation was regarded as an abnormal reaction of the heart to physical effort and, as cardiac enlargement was demonstrated more frequently around the turn of the century when participation in athletics became widespread, the idea of an adverse effect on the heart found wide acceptance. At that time the expression "athlete's heart" was coined to designate a heart damaged by sports. Even before the roentgenologic era, but increasingly after such studies were widely employed, very considerable cardiac enlargement after physical exertion was frequently noted without any signs of myocardial weakness.

These facts made it necessary to study radiographically the influence of continued physical exertion on cardiac size in large series of cases. Many of these investigations leave much to be desired in respect to exactitude as they were based merely on single cardiac measurements and their relation to one or another dimension of the body, nevertheless, they have much value since the resultant errors were largely cancelled by the enormous size of the series

The first extensive studies were conducted by Schieffer. He determined Tr_{H}, Hl, and Hfl in soldiers at the time of induction and after training and showed that cardiac measurements increased in over one half of them These enlargements had no fixed relation to changes of body weight, they were noted when the weight remained steady or even decreased Schieffer also found the heart of cyclists larger on the average than of noncyclists, an observation soon confirmed by Dietlen and Moritz Since most subjects whose hearts enlarged had no complaints and no objective circulatory disturbances but remained perfectly capable of performing exercise. Schieffer properly concluded that the healthy heart could enlarge from exertion

In some cases the enlargement exceeded the average value. Since simultaneous auscultation yielded striking findings and since the capacity for exercise diminished in some of these individuals, Schieffer and Dietlen concluded that exertion could produce dilatation which was no longer physiologic but represented a considerable reduction of cardiac reserve By forbearance from exercise this dilatation disappeared more or less completely.

The first World War also offered an opportunity on a vast scale to test the influence of long lasting, above-average exertion on the heart. All investigators reached the same conclusion under these conditions, more or less marked enlargement of the heart is very common and, furthermore, without any signs of cardiac incompetence Thus, Maase and Zondek found that infantery soldiers after long marches had larger hearts than those who did not march. In the war, Wenckebach also found the heart enlarged in 47 of 100 infantery soldiers The most exhaustive studies are those of R Kaufmann Although his roentgenologic observations are limited to the determination of the diagonal cardiac diameter and to striking changes of cardiac shape without reports on body weight and height, the critical sifting of vast material by an excellent observer makes them extremely valuable. Among soldiers at the front, Kaufmann found many who had enlarged hearts in addition to those with hearts of normal size and definitely small ones. Since these enlargements often receded after rest and suitable treatment, he believed they must be considered primarily as dilatations. He thought this likely since they were more common in

In this connection it would be well to study the normalization of the vectorcardiogram (Schellong) observed by Ickert in "exhaustion" after single physical acts; this tends to be associated with less or no reduction of diastolic blood pressure and is not accompanied by increased size of the heart Since Hochrein conceives of this phenomenon as a sign of "neurocirculatory dystonia," it might be that precisely these cases are the ones in which increased cardiac size depends upon better filling.

Undoubtedly some acute cardiac enlargements after single efforts may persist for hours and days without being pathologic The decision as to whether or not dilatation is pathologic is not easy in the individual case Great caution is in order. Such hearts should be watched closely and examined repeatedly to ascertain how they behave with *carefully arranged added exercise (Rautmann). A relative myocardial* insufficiency would seem very probable when enlargement occurs in a heart

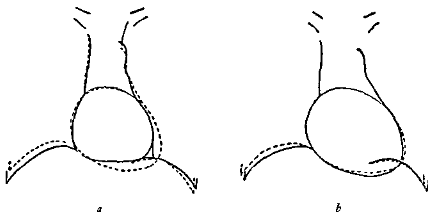


FIG. 60—Increase of vertical heart size after swimming practice in a slender, somewhat underweight youth (*a*) Erect position ———before practice cardiac volume, 368 cc., -- -- after practice, 419 cc (*b*) Recumbent position ———before practice cardiac volume, 579 cc ------after practice cardiac volume 581 cc Youth, 14 years old Height 168 cm, weight 51 5 Kg

normally or excessively large before exercise, when the enlargement progressively increases with continuation of the exercise, so that cardiac size is ultimately abnormally large, and when subjective complaints, pathologic changes in the electrocardiogram, or objective phenomena appear to indicate the onset of circulatory failure

3. *Alterations of the Cardiac Roentgenogram from Continued Physical Exertion Including Participation in Sports*

The alterations of the heart following continued exertion (heavy labor, participation in sports) have been studied with different results. If we are clearer about the subject at present, this is largely due to roentgenology. In the numerous debates on the significance of these alterations, roentgenology made real contributions

It has long been known that wild animals usually have larger and heavier hearts than closely related domesticated species, moreover, long-lasting exercise in animals produces enlargement and increased weight of the heart without any sign of failure. Nevertheless, the opinion prevailed for a long time that, in man at least, strenuous activity normally did not provoke cardiac hypertrophy and, even less, cardiac en-

necessarily have the largest and strongest hearts. The healthy hearts of individuals engaged in strenuous athletics, on the average, are not much larger or heavier than those of the long distance runner as one might have expected from the powerful skeletal muscle development of the former. V Weizsacker explained this apparent contradiction as follows the demands on the skeletal muscles and the heart are entirely different in performances of strength and in continued effort. In sports involving brief performance of strength and tension, sudden tension exerts a marked growth stimulus on skeletal muscles and results in their massive hypertrophy. On the contrary, in sports with continued performance and with less effort per unit of time, as in long distance running, and which, apart from the final dash, never call for maximal expenditure at given moments, increased tension, the growth stimulus for skeletal muscles, is relatively small and therefore induces little hypertrophy. Precisely in these cases, however, demands on the heart are extraordinary, certainly they are decidedly greater than in the short lasting efforts, for example, of a weight lifter These remarks should suffice to indicate that no definite parallelism between the development of skeletal and cardiac muscle can be expected.

Systematic roentgen investigations of cardiac size in perfectly sound, active athletes show that under otherwise equal conditions, the hearts of athletes and short distance runners, on the average, are smallest, those of long distance runners, skiers, football players, and boxers larger, and finally those of bicyclists are the largest; moreover, certain differences exist in respect to the shape of the heart.

To understand changes of cardiac size and shape in athletes, the division of sports into those which make use of tension-strength and those which call for endurance with less expenditure of energy per unit of time, is very fruitful.

Let us first consider sports like shot-putting, weight lifting, or short distance running in which maximal strength is exerted for a short period by sudden tension of certain muscles These performances are associated with increased intra-arterial pressure which means more work for the left ventricle. The ventricle can do this by stronger systolic contractions which finally leads to its hypertrophy (p. 137). This thickening is expressed roentgenologically at most by greater rounding of the left ventricular arc but no enlargement of the cardiac shadow in any dimension (p. 149) However, the possibility of overcoming heightened resistance by increased contraction and pure hypertrophy is limited As a rule it eventuates in dilatation which is not an expression of ventricular incompetence, it is a physiologic method of the normally functioning chamber for overcoming higher resistance by greater diastolic tension of its fibers (Straub) The remarks on page 138 show that this dilatation from increased resistance does not widen the ventricle but leads to practically pure elongation (Moritz) beginning and most marked at the end of its outflow tract, progressing toward the apex, and gradually extending to the inflow tract (Kirch) As explained on page 138, this dilatation from increased resistance and associated left ventricular hypertrophy does not increase the transverse diameter of the cardiac shadow radiographically but only elongates the left ventricular arc, making it rounder and displacing the apex downward and a little outward.

The heart of an athlete well trained for strenuous sports and that of the short distance runner actually shows the alterations one would expect from these general rules for work of the left ventricle against heightened resistance the heart is not

individuals who had had infectious diseases (rheumatic fever, malaria, dysentery, typhus or pneumonia), severe mental excitement, or had suffered from lues or atheromatosis, or were young and physically untrained. Like Schieffer, he also noted that individuals who had followed light occupations or sedentary ways of life as well as those with pendulous hearts often manifested this enlargement after several weeks of military duty For these reasons Kaufmann concluded that some latent damage or functional, constitutional, or even conditional inferiority of the myocardium, as well as nervous influences, played a great role in the occurrence of this enlargement

To Kaufmann, persistent cardiac enlargement indicated that the heart was no longer able to meet increased demands by augmenting its power of contraction, now it must utilize increased tension. Such enlarged hearts seemed less efficient since the margin narrows between the rest volume and the volume enabling the heart to accomplish added work.

Contrary to these conclusions were those of Henschen he stated that the "athletic heart" is not a damaged heart and that this heart "wins the prize " Tung, Hsieh, Bien, and Dieuaide also found enlarged hearts in 45 per cent of completely healthy rickshaw boys Moreover, Kaufmann also observed that these hearts show strong, slow pulsations on the fluoroscopic screen. This observation was confirmed by Rautmann and was supported physiologically by Rein, it speaks against the notion that these enlargements are always pathologic responses It becomes highly probable that enlargements observed after long lasting exertion are not identical and certainly *are very complex in nature (see below)*

In the question of the physiologic effect of prolonged exertion on cardiac size, roentgenologic studies on athletes proved more instructive than those on soldiers and laborers With athletes one starts with material which has been fairly well sifted, it is *composed of physically qualified individuals systematically trained for definite tasks* under good external conditions, soldiers and laborers include fairly large numbers who are not completely sound for constitutional reasons, from previous disease or poor nutrition, and often they have had very serious psychic stress

A special advantage of investigations on athletes is that types of work can be graded to a certain extent and the different burdens placed on the heart can be defined rather *well One can study the reaction of the heart to an exercise which primarily* requires blood volume in excess of normal, to work to overcome elevated peripheral resistance, or to loads predominantly on the left ventricle or on both halves of the heart

All observers who have conducted such investigations agree that sports can lead to enlargement and altered shape of the heart (Dietlen, Moritz, Schieffer, Dedichen, Herxheimer, Rautmann, Bruns, Gotthardt, Bramwell and Ellis, Berger and Olloz, Moritz, Deutsch and Kauf, Reindell, Klaus, Zdansky, and others) Most authors regard the increase in size and shape of the heart as the physiologic result of augmented cardiac work.

Actually this is a manifestation of cardiac adaptation for there is an unmistakable connection between *the average amount of enlargement and the exercise. Only at* the outset does it seem contradictory for the development of skeletal muscle not to parallel heart size or muscle mass, in other words, the strongest individuals do not

heightened diastolic tension of their fibers (p 143) and in its further course leads to hypertrophy of their walls. Since both ventricles are subjected to the same conditions when added blood flows into the heart, both dilate due to increased filling but the right ventricle, with its weaker muscle, dilates more than the powerful left ventricle (Reindell). This dilatation from increased filling is not restricted simply to the ventricles but naturally affects the atria as well (Kirch, v. Braunbehrens and Reindell, Zdansky) The roentgenologic demonstration of atrial enlargement need not imply a retrograde stasis of blood from the ventricle, that is, that ventricular failure exists.

The effects of dilatation from greater filling of both ventricles on the roentgenogram are evident from the discussion in Chapter Three. Elongation of the inflow tract of the right ventricle, corresponding to its transverse course, must broaden the cardiac shadow transversely, while enlargement of the left ventricle, whose inflow tract runs almost dorsoventrad, widens the cardiac shadow in an a-p diameter. Transverse ventricular dilatation, constantly present with dilatation from increased filling, increases cardiac volume on all sides and causes greater rounding of the left heart border. A glance at figure 65a, b, and c will visualize these results. It is an interesting, although still unexplained, physiologic fact that the adaptive enlargement of the athletic heart persists during rest, that is, even when the filling pressure and the stroke volume of the heart are not increased. This cardiac enlargement is not utilized functionally at rest but is evoked to augment stroke volume during physical exertion. Reindell designates this as an altered diastolic orientation of the myocardium, a "regulative cardiac dilatation," and ascribes it to altered elastic properties of the myocardium, conditioned by nervous and hormonal factors Probably a tonic orientation of the myocardium, regulated by vegetative and hormonal factors and independent of the degree of mural thickening plays a role (Zdansky), this should have great influence on cardiac performance

The changes in size and shape of the heart in those indulging in long lasting sports and in those expending less energy per unit of time correspond perfectly with the situations depicted. The heart is more or less universally enlarged with bulging borders and a rounded apex. The cardiac waist is preserved or even deepened owing to prominence of the left cardiac border.

The largest hearts with bilateral enlargement and particularly marked elongation and rounding of the left ventricular arc are found in cyclists This is easy to understand since cycling is a prolonged event with expenditure of energy in terms of distance per unit of time but is combined with tension-strength, constantly called into action in spurts and in climbing slopes. Cycling places the highest demands on the heart.

The size and shape as well as pulsations of the heart show some changes from athletic effort. Apart from the resting bradycardia of the trained athlete, certain changes in the form of ventricular pulsations are visible roentgenologically. The notches of the ventricular roentgenkymogram are blunt, more convex or show lateral plateaus (Reindell). These changes somewhat resemble those observed in myocardial injury. The demonstrable changes of breadth of pulsations in different parts of the left ventricular arc are also striking While in most normal hearts the excursions of the left ventricular arc near the apex are larger than those near the base, with increas-

widened but merely gives a hint of an aortic configuration by elongation and more rounding of the left ventricular arc (fig. 70) Since not only the outflow tract of the left ventricle underlying the left ventricular arc but also its inflow tract is more or less lengthened, there is greater projection of the cardiac shadow into the vertebral shadow in the left anterior oblique position; in the right anterior oblique position the longitudinal diameter is lengthened. To understand these alterations, it is advisable to glance at figure 65a, b, and c, in which the projection of the in- and outflow tracts of the ventricles is shown in the three major positions.

Frequently, if one finds a large or very large heart in many athletes, the abuse of alcohol or nicotine and occasionally lues may also play a role (Romberg) One might also consider that among those indulging in the strenuous sports, many are in the fourth and fifth decades so that the manifestations of coronary insufficiency without exogenous injuries should be borne in mind

The situation is entirely different with long distance runners, skiers, swimmers, boxers, that is, in sports in which training is for long distance performance and for prolonged exertion. All investigators agree that these athletes have striking enlargement of the cardiac shadow which is not limited to the longitudinal dimension but also involves the transverse. Roentgenologic analysis reveals that this enlargement affects both halves of the heart and especially the right

This bilateral enlargement is explained by the special hemodynamic conditions existing in these sports In these long lasting performances with less expenditure of energy for distance per unit of time, work is executed with economic distribution of reserve forces, with simultaneous deepening of respiration, with many groups of muscles moving steadily and consequently increasing their O_2 requirements enormously The satisfaction of O_2 demand is possible only by mobilizing all reserves of blood, by excluding certain capillary beds, as well as by increasing the circulating blood volume greatly over its basal value In this way the heart receives in a given unit of time, a volume of blood for propulsion which is considerably greater than the resting value It fulfills this task partly by the Bainbridge reflex, this is released by higher pressure in the right atrium and leads to cardiac acceleration, also partly responsible is augmented accelerator tonus which releases more powerful and more frequent contractions and more complete emptying of the ventricles to produce a larger stroke and minute volume, to a great extent, however, the effect is attained by diastolic ventricular dilatation This "dilatation due to increased filling" (Zdansky) is the direct result of subjecting the heart to a filling pressure above normal This is not only hemodynamically important but also possesses special interest because it offers a key to understanding the cardiac enlargement so common in sports of this kind

As explained on page 143, increased diastolic filling pressure leads to dilatation of the ventricles in a longitudinal and transverse direction. It begins and is most marked in the parts of the ventricles that receive additional blood streaming into them in diastolic relaxation (Zdansky). With increased influx of blood from the atria, dilatation and elongation begin in the domain of the inflow tract, progress toward the apex, and finally involve the outflow tract. This dilatation may be considered a physiologic reaction of the normal myocardium and is not a sign of failure. It constitutes a prerequisite for the ventricles to eject larger quantities of blood by

myocardial damage. Conduction time, often prolonged, and the resting bradycardia of the athlete is regarded by Rein as augmented vagal inhibition; this vanishes immediately in exercise tests to give way to normal conduction time and rate. Likewise the electrocardiographic changes (Schlomka, Reindell, and others) described as alterations of the atrial and of the QRS complex as well as of the terminal deflection do not indicate any pathologic change in the myocardium.

The most obvious change in the athletic heart is its enlargement and change of shape In single cases no relation exists between the type and duration of athletic participation on the one hand, and increased cardiac volume on the other Remarkable individual deviations from average values are observed in well trained athletes with outstanding records and with either very large or definitely small hearts. Naturally, this raises an important problem in respect to "athlete's heart" There must be a reason why one person requires an obvious cardiac enlargement to accomplish a task while another has a practically normal or even small heart.

Theoretically there are two possibilities to explain these individual differences in response. First, different people employ different technics of muscle action and different technics of respiration to attain the same objective and this may result in different work for the heart. This assumption is improbable from the start, every well coordinated bodily performance is guided chiefly by subcortical reflexes which are subject only to minor individual differences Moreover, in training, every athlete follows procedures so similar that differences of practical importance to all intents and purposes are excluded.

There remains the second possibility individual differences of the myocardium or vegetative nervous system are responsible for the diverse reactions of the heart. At present very little is known about differences undoubtedly existing in the functional efficiency of normal skeletal and heart muscle. Whether this depends upon different metabolic processes in the muscle cell or an anatomically established or functionally conditioned blood supply of the muscle, is unknown. We are not much better informed on how far vegetative influences act directly or through hormones on the vessels or muscle fibers to affect the work reaction of the muscles in various ways It is, at least, highly probable that such influences exist and account for the diversity of cardiac reactions in athletics. In this connection the studies of W Raab have special interest. Raab showed by chemical methods on man and animals how adrenalin and other sympatheticommetic amines, partly precursors and partly oxidation products of epinephrine, which are formed in the adrenal medulla, in the myocardium, and in the vessel walls, are electively stored and accumulate in the myocardium These materials participate decidedly in the occurrence of myocardial hypertrophy and dilatation, if formed or stored excessively in the heart, they can damage it Raab was able to determine a critical concentration of these materials for the heart beyond which cardiac death promptly occurred. The formation of these materials was stimulated by exertion, cold, and mental excitement Their storage in the heart was potentiated by thyroid hormone and was regulated by the steroids of the adrenal cortex and gonads (Raab).

Considering that the discharge of these extremely cardioactive substances from the adrenals and the vessels and their autochthonous formation in the myocardium is subject to vegetative regulation and that the action of these substances can be

ing enlargement of the athlete's heart the reverse is more common at rest; the pulsations near the apex are smaller than those in the basal section of the left ventricle Schwarz observed this long ago during fluoroscopy when the heart showed left sided hypertrophy and dilatation On the basis of roentgenkymographic studies, Stumpf contrasted these pulsations as types I and II and drew far reaching conclusions in respect to the state of the myocardium (p. 53). Reindell explains the small pulsations near the apex of enlarged hearts of athletes as follows the basal parts participate predominantly in systolic emptying of the ventricle while the area near the apex stores larger amounts of residual blood. Actually, after exercise, when the heart contracts more strongly under the influence of augmented accelerator tonus, type II tends to be converted into type I because of more complete emptying of residual blood stored in the apical parts of the ventricles With myocardial damage this conversion usually fails to take place, this could be compatible with defective ability of the ventricle to contract and empty Likewise the convex and plateau-shaped ventricular curves just mentioned usually give way to normally pointed pulsations in the sound heart of an athlete during exercise while they often fail to change if the heart is damaged (Reindell).

In regard to the time required for the occurrence of cardiac enlargement and change of shape, valid generalizations cannot be made. Among other things it depends upon the extent of demands placed upon the heart Within the limits peculiar to the sport, age seems to play no essential role, certainly the youthful heart is not, apparently, more susceptible to enlargement. The enlargement develops very insidiously After participation in sport for one or two years, universally enlarged hearts are found as well as hearts of normal size. Increase of volume occurs even at the beginning of athletic participation in slender asthenic youths and often depends upon better distribution of blood (p 103), this, in turn, is conditioned by increased tonus of the skeletal muscles and the peripheral vessels (Zdansky). In general, with longer participation in sports, the number of definitely enlarged hearts increases.

When athletic activity ends, cardiac enlargement and transformation gradually recede but this regression is not always complete.

This does not hold for acute enlargements repeatedly observed in connection with intense athletic effort Usually these are relative overstrains and occasionally are announced by a decline in efficiency (Reindell) although often objective evidence of such decline cannot be secured These acute enlargements usually recede after a few weeks of rest. They suggest a transient myocardial insufficiency from overstrain which may produce characteristic changes in the electrocardiogram after some burden is imposed (Reindell). In athletes in the fourth and fifth decades, dilatation appears more frequently (Deutsch and Kauf). The extent to which an intercurrent myocarditis or a disturbance of coronary perfusion is causal must be carefully determined in each acute cardiac enlargement Strict avoidance of exertion and greatest care on resumption of the sport is definitely indicated.

In the domain of the pathologic, patients with goiter often show just as much cardiac enlargement as those participating in strenuous athletics.

If we dismiss cardiac enlargement which is definitely pathologic, increased cardiac size in athletes, even when very conspicuous, still may be entirely normal. Supporting this is the absence of any electrocardiographic changes indicative of

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If we dismiss cardiac enlargement which is definitely pathologic, increased cardiac size in athletes, even when very conspicuous, still may be entirely normal Supporting this is the absence of any electrocardiographic changes indicative of

fiber length; then, enlargement becomes an adaptive phenomenon. Whether this adaptation is physiologic in a given case, a vegetative-hormonal overdemand on the heart muscle, or a pathologically diminished efficiency (myocardial incompetence), cannot be decided definitely by radiographic methods. An exclusive or almost exclusive elongation of the cardiac shadow with normal width makes myocardial failure highly improbable (Rautmann, Gotthardt, Eimer); predominant transverse enlargement awakens the suspicion of reduced myocardial efficiency These conclusions are, however, by no means absolute The greatest difficulties are presented, as always, by borderline cases and it is never permissible to make a decision solely on the basis of the roentgenogram. Sudden cardiac enlargement in the course of exertion or gradual, progressive enlargement must always be regarded as probably pathologic

Since any crude morphologic cardiac finding has equivocal meaning, the result of roentgen examinations in single cases can be evaluated only in connection with the entire clinical situation. Within this frame it may have great significance. At all events, the observation of alterations of cardiac size and shape after a single or long lasting, graded exercise represents a valuable supplementation and perfection of tests of cardiac function

XIII. Roentgenologic Tests of Cardiac Function

The demonstration of alterations experienced under the influence of definite added exertion awakened the hope that roentgenology might serve to test cardiac function.

Investigations of the alterations in diastolic cardiac volume (p. 117) after standardized exercise was undertaken with this in mind After exercise (bending the knees, climbing stairs) incompetent hearts may enlarge (p. 119). Naturally this enlargement does not prove myocardial incompetence The normal heart can also enlarge after exertion from an interplay of peripheral circulatory regulations and other extracardiac factors Thus, the small hearts of asthenic, physically untrained individuals, labile from a circulatory standpoint, particularly enlarge from greater influx of blood after exertion (p 117) Consequently, increased cardiac volume after single exercises suggests myocardial incompetence only when the heart was previously normal in size or enlarged

It was hoped that roentgenologic tests of cardiac function would be refined by roentgenkymographic registration of pulsation size and determination of the systolic and diastolic cardiac diameters before and after exercise (running, knee bending) (Stumpf, v. Braunbehrens and Reindell, Teschendorf, Kienle) Normal hearts which become smaller after exercise show larger pulsations and the systolic cardiac diameter diminishes more than the diastolic. This happens because stroke volume increases and systolic emptying is more complete With incompetent hearts, on the contrary, exercise either does not affect size or causes enlargement while pulsations along the left cardiac border become smaller The reduction of the excursions is characterized by increase of the systolic cardiac diameter while the diastolic remains unaltered or both the systolic and diastolic diameters increase, the first more than

increased by hormonal effects and exogenous factors, perhaps we have a key for understanding the differences of reaction in the hearts of athletes, the extent of which is so individually variable. Certainly these interweaving factors are extremely complicated even under physiologic conditions and undoubtedly by collaboration they can produce results which gradually differ from case to case If this happens in daily life, certainly it would hold to a greater extent in sports which place the utmost demands on the heart and circulation from a physical and emotional standpoint With such extreme conditions it would be easy to understand how reactions can appear in the heart which stand at the borderline of the physiologic and at times pass into the domain of the pathologic.

Sudden deaths while swimming are by no means rare and no definite anatomic lesion is found To explain them, recourse has been made to most diverse hypotheses, to cold allergy for example Perhaps an excess above the critical myocardial concentration of the substances described by Raab could be responsible since these agents may be discharged in greater quantities by the stimulus of cold

Irrespective of the significance of Raab's findings, taken in conjunction with the individually variable increase of cardiac size under the influence of athletics, they indicate that the reaction of the heart to sports still conceals unsolved problems. It is certainly not absolutely established that cardiac enlargement, which is often obvious, should be dismissed as a completely harmless reaction to physical exertion even though the electrocardiogram is normal and the capacity for physical performance is excellent This is problematic because these enlargements tend to appear precisely in sports placing demands on the heart and circulation vastly exceeding the limits to which man is exposed under natural conditions These performances are no longer physiologic, consequently they may lead to nonphysiologic effects on the heart At all events, an appropriateness of athletic cardiac enlargement does not prove it is completely innocuous Rather, it raises the question What becomes of these enlarged hearts? What are their prospects for life? These questions can be answered only on the basis of exact roentgenologic determinations of cardiac size for many years in a large number of athletes Certainly roentgenology can contribute much to the solution of these problems

It seems that the great individual differences in the reaction of cardiac size to sports opens the view to certain constitutional differences in the circulatory org ns and in the vegetative and hormonal regulation of circulation whose ultimate significance can be evaluated only after many years

In summary, serial roentgen examination reveals that persistent exertion in general tends to cause enlargement of the heart and to change its shape, frequently this is associated with an altered type of pulsation Many hearts, however, remain completely unaltered and some, though definitely small, may meet the utmost demands The extent of enlargement depends upon the type of activity but is partly determined by constitutional and conditional properties of the myocardium, of the peripheral circulation, and of the vegetative and centrally regulated hormonal system. Cardiac enlargement does not always have the same meaning. Improved distribution and augmented flow of blood may produce it, this certainly happens in many young, ptotic-asthenic individuals and in those with pendulous hearts It may also indicate that the heart is unable to discharge increased loads with its original

fiber length, then, enlargement becomes an adaptive phenomenon. Whether this adaptation is physiologic in a given case, a vegetative-hormonal overdemand on the heart muscle, or a pathologically diminished efficiency (myocardial incompetence), cannot be decided definitely by radiographic methods An exclusive or almost exclusive elongation of the cardiac shadow with normal width makes myocardial failure highly improbable (Rautmann, Gotthardt, Eimer), predominant transverse enlargement awakens the suspicion of reduced myocardial efficiency. These conclusions are, however, by no means absolute. The greatest difficulties are presented, as always, by borderline cases and it is never permissible to make a decision solely on the basis of the roentgenogram. Sudden cardiac enlargement in the course of exertion or gradual, progressive enlargement must always be regarded as probably pathologic

Since any crude morphologic cardiac finding has equivocal meaning, the result of roentgen examinations in single cases can be evaluated only in connection with the entire clinical situation Within this frame it may have great significance. At all events, the observation of alterations of cardiac size and shape after a single or long lasting, graded exercise represents a valuable supplementation and perfection of tests of cardiac function

XIII. Roentgenologic Tests of Cardiac Function

The demonstration of alterations experienced under the influence of definite added exertion awakened the hope that roentgenology might serve to test cardiac function

Investigations of the alterations in diastolic cardiac volume (p. 117) after standardized exercise was undertaken with this in mind After exercise (bending the knees, climbing stairs) incompetent hearts may enlarge (p 119). Naturally this enlargement does not prove myocardial incompetence. The normal heart can also enlarge after exertion from an interplay of peripheral circulatory regulations and other extracardiac factors Thus, the small hearts of asthenic, physically untrained individuals, labile from a circulatory standpoint, particularly enlarge from greater influx of blood after exertion (p 117). Consequently, increased cardiac volume after single exercises suggests myocardial incompetence only when the heart was previously normal in size or enlarged

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the last Both reflect a smaller stroke volume of the incompetent heart as systolic emptying of the ventricles become impaired.

Moreover, the alterations in the type of pulsations along the left cardiac border (p 53) combined with changes in cardiac size after exercise has been studied in detail According to Stumpf, a conversion from type I to II (p 54) with fixed or increasing cardiac size favors cardiac incompetence (usually coronary insufficiency). a conversion of type II into I with increase of cardiac size, slower rate, and lateral plateau formation of the ventricular curve indicates "reduced performance" in decompensated lesions and hypertension, finally increase of cardiac size, pathologic changes in the curves and decrease of rate should indicate cardiac failure from coronary disease or myocardial damage. Reduction of heart size after effort with unchanged type I or a conversion of type II into I with a simultaneous rise in rate, often present and frequently observed with athlete's heart (p 54), usually indicates good function

Another functional test is based on change in heart size and pulsations with a "graded" Valsalva test (Burger) before and after exercise with simultaneous measurement of the blood pressure, pulse rate, electrocardiogram, and vital capacity.

Teschendorf distinguishes five groups according to the outcome of the test

1 Bilateral reduction of heart size from Valsalva before and after exercise with normal hearts and "well compensated cardiac disease"

2 Bilateral reduction before, uni- or bilateral reduction after exercise in which a "dilated hypertrophied or abnormal myocardium cannot adapt itself to the reduced inflow of blood Teschendorf includes in this type the hypertrophied athlete's heart and considers this outcome of the test a "warning signal"

3 No reduction of cardiac size on one side, on the left in left sided hypertrophy without "tendency to insufficiency" and on the right usually with "stasis in the lesser circuit and in the right heart

4 No reduction of cardiac size before and after exercise is "constantly a sign of markedly reduced functional capacity, often associated with a very unfavorable prognosis"

5 Slight reduction of heart size before and marked reduction after exercise "usually indicates relaxed hearts which still respond to the stimulus of exercise"

At present such studies lack widespread approval since the test, in part, merely confirms clinical, roentgenologic, and electrocardiographic studies, moreover, the Valsalva test yields noncharacteristic and equivocal results which contain numerous sources of error (p 112) In addition the performance of the Valsalva test in many patients with hypertension, myomalacia, mitral stenosis, heart block, and cardiac decompensation is not devoid of danger.

XIV. The Influence of the Introduction and Loss of Fluids on Cardiac Size

Severe acute blood loss as well as dehydration from thirst, uncontrollable vomiting or profuse diarrhea (fig. 61) reduces cardiac size (Schieffer, Meyer, Assmann). After spontaneous filling of the vascular system or transfusions, the cardiac shadow rapidly returns to its original size (Meyer and Seyderhelm) In experimental animals the previously normal heart is enlarged by transfusions and this parallels the rise of

venous pressure (Meek and Eyster) The simultaneous increase of the visible pulsations of the heart indicates a greater stroke volume. Only with venous pressures above 150 mm. H_2O (in rabbits) was there no further increase of heart size, on the contrary, the pulse rate rises distinctly

Increases of cardiac size are also observed in man after transfusions Although very transient (fig. 62) they indicate that plethora can provoke enlargement (Meyer).

As a rule a single venesection causes no or, at most, a transient reduction of cardiac size (Dietlen, Rosler). Nevertheless Grothusen as well as Tschilow and Christoff demonstrated in about one half of their patients a reduction of heart size about seven hours after venesection although the amount of blood removed did not

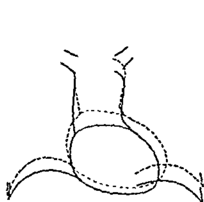


Fig 61

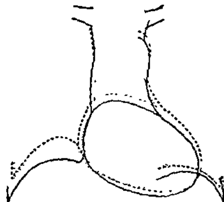


Fig 62

Fig 61 —Reduction of cardiac size from copious diarrhea in acute gastroenteritis ——— at height of disease ... ..after diarrhoea ceased (two days later). Besides the elevation of the diaphragm, considerable enlargement of the heart is apparent (horizontal orthodiagram).

Fig 62 —Transient cardiac enlargement after intravenous injection of a large amount of fluid ——— before the injection volume = 515 cc , ------ seven minutes after the injection of 1 liter of normosal volume = 580 cc , forty-five minutes after injection volume = 494 cc.

exceed 180 to 300 cc. Since most patients were decompensated, improved circulation accompanying diuresis or myocardial improvement itself could have led to the reduction of cardiac size The withdrawal of so little blood is not directly noticeable, at most, it could lead indirectly to a smaller size of the heart

Repeated loss of blood leading to severe anemia can produce gradual cardiac enlargement in experimental animals after a preceding reduction in size, this is probably due to anemic myocardial incompetence (p. 213).

The reduction of heart size after copious diuresis will be discussed in another place.

XV. The Influence of Rate on Cardiac Size

Purely hemodynamically, increased rate must reduce cardiac size (Barcroft) and slowing lead to cardiac enlargement since the extent of cardiac filling, under other-

wise equal conditions, depends upon the length of diastole. If this is short, filling is less and the stroke volume smaller, if long, filling of the heart and its stroke volume become greater.

Naturally slight differences in rate do not obviously affect the roentgenologic size of the heart. Animal experiments (Meek) indicate that moderate acceleration provokes only a trifling decrease in size and not until the rate exceeds 110 does the heart rapidly become smaller, then, diastole in which filling is rapid and under high pressure, shortens. In investigations on man in the horizontal prone position, cardiac size need not diminish despite a tachycardia up to 120 per minute induced by atropine nor is it essentially less than on standing (Nylin, Larsson, Kjellberg)

Moritz, and Hodges and Eyster as well as Scherf and Zdansky observed reduction of cardiac size in erect men during atropine tachycardia, in general this became more distinct as the rate increased. Scherf and Zdansky also found a similar effect after the intravenous injection of adrenalin although sometimes the blood pressure rose above 200 mm Hg In these cases reduction of cardiac size was related to increased rate and also to the positive inotropic effect of adrenalin on the myocardium. Even here, several factors are active, some support the size-reducing effect of acceleration and others diminish it. The same observers as well as Brams and Strauss noted a reduction of cardiac size after the inhalation of amyl nitrite. This is a result not only of increased rate but probably an effect on the accelerators and certainly upon peripheral vasodilation which diminishes return of blood to the heart

Clinical experiences are in agreement with these experiments. A smaller cardiac shadow is frequently observed in fast tachycardias (Dietlen, van Zwaluwenburg), enlargement is common with bradycardia, especially in complete heart block. The opposite behavior of cardiac size is almost as common. In human pathology and physiology, in addition to the higher rate, complicating factors very often weaken or even reverse the effect of the pulse rate on cardiac size. Above all, the state of the myocardium decisively influences changes in cardiac size with varying rates Bradycardia as well as tachycardia may be a manifestation of myocardial injury, moreover, an increase of rate, by creating a disproportion between the work imposed on the heart and coronary perfusion may lead to hypoxia and consequently to insufficiency and dilatation while a decrease in rate may improve perfusion and aid in myocardial recovery

Accordingly, in paroxysmal tachycardias, after an initial reduction of size, the heart occasionally enlarges and this is ascribed to cardiac failure (Kahlstorf). Both halves of the heart dilate equally and cause the cardiac shadow to dilate like a balloon Only under abnormal working conditions of the heart does the dilatation primarily affect those parts which must perform the greatest tasks; in other words, in hypertension dilatation affects the left half predominantly and with emphysema the right heart dilates most.

Even considerable dilatation appearing in the course of paroxysmal tachycardia may vanish completely as normal rate returns and then reappear with recurrent cardiac acceleration (fig. 63). Diminution of cardiac size was also striking in a

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Cardiac enlargement may be associated with a reduction in width of the vascular band (Kahlstorf) when filling of the great vessels decreases or with widening of the band (fig. 63) with stasis in the great veins.

Long lasting tachycardias can lead to cardiac dilatation which no longer recedes even when the rate returns to normal. On the other hand, we observed reduction of size of a dilated heart after subtotal thyroidectomy although the rate did not decrease. Such observations clearly emphasize how complicated the situation is; they reflect the great influence which the *myocardial status* and *mode of myocardial reaction* exerts in alterations of cardiac size, this also brings many apparent paradoxes closer to understanding.

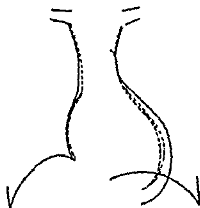


Fig. 63



Fig. 64

Fig. 63.—Cardiac enlargement from paroxysmal tachycardia. Male, 22 years old. ——— October 16, 1933 tachycardia for some days, . . . October 20, 1933 normal rate, - - - October 21, 1933 one hour after beginning of new attack, . . . October 24, 1933 *normal rate*. Note the widening of the superior vena caval shadow on Oct. 16 *from retrograde stasis due to cardiac failure*.

Fig. 64.—Reduction of cardiac size after the transition of paroxysmal ventricular tachycardia with a rate of 144 into complete heart block with rate of 54. (——— December 27, 1934 rate, 144 per minute, ... December 31, 1934 rate 54 per minute.)

Attinger had occasion to observe ventricular fibrillation radiographically. Without reporting on heart size he depicted the pulsations appearing on the fluoroscopic screen. Besides the total arrhythmia, he perceived "fine" movements on the left ventricular arc which created the impression that each point was "*in the grip of its own vibration and twitching.*" The author considered these fine movements as contractions of parts of the left ventricle interpolated between *total contractions*.

XVI. The Effect of Arteriovenous Shunts on the Heart, Exclusive of Persistent Ductus Arteriosus

Abnormal communications between the large peripheral arteries and veins may be congenital or acquired. Usually the acquired ones follow trauma (*projectiles*,

wise equal conditions, depends upon the length of diastole. If this is short, filling is less and the stroke volume smaller, if long, filling of the heart and its stroke volume become greater

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Even considerable dilatation appearing in the course of paroxysmal tachycardia may vanish completely as normal rate returns and then reappear with recurrent cardiac acceleration (fig 63). Diminution of cardiac size was also striking in a patient when the rate of 144 was replaced by complete heart block and an unusually high ventricular rate of 54 The bradycardia tended to reduce cardiac size by diminishing work and improving coronary blood supply (fig. 64).

occasionally better transparency of the previously dark lung fields (Weber, Frey, Eppinger, Rieder, Fick, McQuire, Puchlew) which reflects the momentary unloading of the heart and lesser circuit Some observers have reported inward sinking of the right cardiac border (Caro). Zdansky saw universal reduction of cardiac size, immediate bradycardia, and simultaneous greater transparency of the lung fields in a woman whose arteriovenous fistula between the Arteria and Vena iliaca comm had been produced by a bomb fragment Her first signs of circulatory failure appeared six months after the injury and gradually increased until ascites, hepatic congestion, edema, and pulmonary congestion occurred.

The idea that blood flowing into a vein under arterial pressure produces greater cardiac filling and causes tachycardia by a Bainbridge reflex must be abandoned. Usually venous pressure increases only when the right heart fails Moreover, in animal experiments interruption of an arteriovenous short-circuit in a lower extremity immediately results in slowing the cardiac rate despite previous ligation of the inferior vena cava, no alteration of cardiac filling pressure could occur (Fick) Since the compression test is still positive when all nerve pathways are interrupted, the assumption of a nervous reflex must likewise be excluded (Fick) The only remaining releasing cause of the compression phenomenon is the mechanical interruption of the abnormally fast stream of blood flowing out of the artery into the vein The manner by which the interruption momentarily corrects this abnormality of the circulatory situation is still unexplained

Permanent interruption of the short-circuit is the only way to relieve the heavy load on the heart After the shunt is removed by operation, all observers note a reduction of cardiac size, decline of the rate and minute volume, reduction of pulse pressure amplitude, rise of the systolic pressure (previously, often low) as well as the disappearance of cardiac murmurs which are often present Even when cardiac dilatation is considerable and associated with pulmonary and hypertensive stasis, it recedes (Ranzi, Bier, Eppinger, Rieder, Laplace, Quattlebaum, Puchlew, v. Oppolzer) Intervention may be lifesaving despite the presence of severe decompensation

Among the congenital arteriovenous short-circuits, arteriovenous aneurysms of the lungs in which the branches of the Art. and Vena pulmonalis communicate directly are of interest in the present connection They are relatively rare In more than one half the cases it is a partial manifestation of Osler's hereditary hemorrhagic telangiectasia Although present in early childhood, it is usually discovered only in later years when clinical manifestations appear Often it represents an incidental roentgen finding since clinically it may be completely asymptomatic. To be sure, cyanosis and clubbing of the fingers and toes are often present since up to 80 per cent of the blood may pass through the shunt. The size of the shunt has, however, no relation to the presence or absence of cyanosis. Ettinger and coworkers observed the appearance of cyanosis and clubbing of the fingers over the course of several years. By rupture of the aneurysmal wall hemoptysis may develop and even prove fatal (Wilkens, Costa, Barnes and Stedem). Owing to the temporal connection of hemoptysis to attacks of unconsciousness, Lindgren considered that the latter could have been produced by cerebral air embolism

Congenital pulmonary arteriovenous aneurysms are not insignificant vascular anomalies With bleeding, their operative removal is indicated. Lindgren and Dorsen-

splinters, stab wounds, or the *effect of blunt force*), *rarely are they of infectious* origin (mycotic). Traumatic arteriovenous short-circuits develop only some time after the injury and the communication can gradually enlarge under the influence of the blood pressure. Different forms of arteriovenous shunts are recognized: (1) arteriovenous fistula, a simple communication between the adherent artery and vein, (2) varix aneurysmaticus in which a varicose dilatation of veins comes under the influence of arterial pressure, (3) aneurysma varicosum in which the vessels are joined through a false aneurysmal sac, (4) aneurysma arteriovenosum in which an arterial aneurysm communicates with a vein.

Arteriography shows that the section lying central to the short-circuit is usually dilated while the peripheral section is narrowed or, occasionally, obliterated (Fick) *In this way one may also demonstrate the more or less extensive development of* collateral circulation. This has practical importance since then ligation of the artery to remove the communication as a rule provokes no nutritional disturbances in the field supplied.

Clinically, arteriovenous short circuits always have importance since aneurysmal dilatation of the venous section may lead to rupture or compression phenomena in vital organs They also have special significance since they can burden the circulation and the heart This does not hold for congenital short-circuits (Reid). However, the more central an acquired communication is located, the greater is the danger of an actual revolution of circulatory relations to create an ominous overload for the heart. The heart does not grow with this load. Sooner or later failure occurs with *hepatic congestion, ascites, edema, pleural effusion, and pulmonary congestion* The flow of blood from artery to vein naturally results in a fall of blood pressure which the heart attempts to restore by increasing the rate and stroke volume (Eppinger, Kisch, and Schwarz). Consequently tachycardia and enlarged stroke volume with reduced diastolic pressure are constant signs of a broad communication between an artery and vein.

Radiographic views show a more or less universal enlargement of the heart with augmented rounding of both borders and occasionally bulging of the pulmonary arc. The remarkably large pulsations indicating the large stroke amplitude are in keeping with the size and the fast rate of the heart. With higher grades of cardiac dilatation the vascular markings of the lungs often increase diffusely and generally the *lung fields are hazy, frequently a right or* bilateral hydrothorax may be demonstrated. The absence of left atrial enlargement permits one to exclude a mitral lesion with great probability although occasionally its presence is assumed clinically when the usual systolic murmur is audible and the second pulmonic sound is accentuated *Moreover, the roentgen findings may closely resemble those of a decompensated* thyrotoxic heart

Of decisive diagnostic significance for the existence of an arteriovenous short-
[illegible] If pressure at the site of the arterio-
ry and vein,
blood pres-
sure (Nicoladoni and Israel) with marked slowing of the rate (Schapiro, Branham) With simultaneous fluoroscopic observation one may often see, besides the impressive slowing of cardiac pulsations, a considerable reduction of cardiac size and

occasionally better transparency of the previously dark lung fields (Weber, Frey, Eppinger, Rieder, Fick, McQuire, Puchlew) which reflects the momentary unloading of the heart and lesser circuit. Some observers have reported inward sinking of the right cardiac border (Caro). Zdansky saw universal reduction of cardiac size, immediate bradycardia, and simultaneous greater transparency of the lung fields in a woman whose arteriovenous fistula between the Arteria and Vena iliaca comm. had been produced by a bomb fragment. Her first signs of circulatory failure appeared six months after the injury and gradually increased until ascites, hepatic congestion, edema, and pulmonary congestion occurred.

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Congenital pulmonary arteriovenous aneurysms are not insignificant vascular anomalies With bleeding, their operative removal is indicated Lindgren and Dussen-

berg reported successful extirpation which led to the disappearance of hemoptysis and cyanosis.

At present relatively few roentgen findings have been published (Smith and Horton, Jones and Thompson, Rundles, Lindgren, Witheaker, Baker and Trounce, Crane and coworkers, Duisenberg and Arismendi, Ettinger and coworkers, Yater and coworkers, Hedvall, Hedinger and coworkers, Schludermann, and others). The author has observed two cases. The rounded, polycyclic or tortuous soft tissue shadows are usually sharply defined although they may have vague contours when hemorrhage has occurred into the surrounding lung parenchyma. The short-circuit shows a preference for the middle and lower lobes but it may occur anywhere in the lungs Occasionally mural calcification is perceptible (Jones and Thompson, Baker and Trounce). Even on scout films, but more distinctly on tomograms and with angiography, one recognizes the abnormal entrance and exit vessels which show a tortuous course and abnormal communications. In an unpublished case of Schlotter, Zdansky observed striking emphysema of the left upper lobe which contained an unusually large, tortuous aneurysm This seemed to indicate an anomalous anlage for this section of the lung.

Only rarely on fluoroscopy are systolic-expansile pulsations of the aneurysmal shadow observed, although apparently a reduction of the shadow can often be demonstrated with the Valsalva test and an increase with Müller's test. The failure of the structure to change its size does not, however, speak against an arteriovenous aneurysm.

In contrast to acquired shunts of the greater circulation and persistent ductus arteriosus, the cardiac shadow is not enlarged On the contrary Lindgren saw enlargement of the previously normal heart after successful removal of the aneurysm, he ascribed this to increased resistance in the lesser circuit after the short-circuit was abolished (?).

In very rare cases an arteriovenous pulmonary aneurysm may be traumatic in origin Castex, di Cio and Battros described the case of a woman who had sustained a penetrating wound of the lung nine years earlier.

—Chapter Three

Atrophy, Hypertrophy, and Dilatation of Single Sections of the Heart

Atrophy, hypertrophy, and dilatation represent reactions forced upon single sections of the heart by changes in their working conditions, in the anatomic status of their musculature, or in certain vegetative or hormonally regulated events. Usually they involve an adaptation phenomenon which is often compensatory because it not only results from altered working conditions but is also a successful or abortive attempt of the myocardium to perform greater work under changed conditions

Myocardial atrophy occurs when activity is reduced. It is observed, for example, in the left ventricle from mitral stenosis or in the entire heart from cachexia and severe nutritional disturbances Anatomically it often shows the features of brown atrophy. Since filling of the heart or of its single sections is reduced in all these conditions, roentgenograms show a more or less distinct reduction of cardiac size. In mitral stenosis left ventricular atrophy is expressed as flattening and abnormally steep descent of the left ventricular arc With myocardial failure an atrophic heart may undergo dilatation.

Myocardial hypertrophy always occurs when the load applied exceeds normal * Pure hypertrophy is relatively rare. In a majority of cases hypertrophy is combined with dilatation although the latter may be very slight. While this holds for the ventricles, it prevails even more for the atria which lack strong muscle. Dilatation of a ventricle is not necessarily a sign of muscular failure for it may be the immediate consequence of the extent and nature of the imposed added work.

Added work for a ventricle may consist of (1) advancing its normal content against heightened resistance, (2) advancing its abnormally large content against

* Occasionally hypertrophy can be vicarious when parts of the muscle die, owing to myocarditis or myomalacia, and are replaced by scar Vicarious hypertrophy of fibers remaining intact may lead to thickening of the surrounding wall (Zdansky), this may be distinct in the roentgenogram although it has not received the attention it merits In general, hypertrophy occurs when demands upon a section of the heart are increased

normal resistance, or (3) advancing its abnormally large content against heightened resistance

The normal ventricles are able to overcome increased resistance by stronger contraction and practically pure hypertrophy. The volume of the ventricles may remain normal Kymographic registration (Reindell) and angiocardiography (Cabrera and Monroy) may even show a diminution of the systolic volume by more complete emptying.

Dilatation from increased resistance clinically occurs very often when the ventricle is unable to discharge its contents against heightened opposition as completely as normally, the increased amount of residual blood retained is added to the normal quantity of blood received from the atrium in the succeeding diastole. Dilatation from increased filling occurs when the ventricle obtains an abnormally large volume of blood from its atrium or when additional blood *returns from its related vessel*, the result is an abnormally large diastolic content of the ventricle.

Dilatation from increased resistance as well as from increased filling enables the ventricle to augment its execution of work precisely to the extent necessary in accord with the Frank-Starling laws, these laws stipulate that the higher the tension of muscle fibers—that is, of their stretch before contraction—the greater the force of contraction of a muscle (under otherwise equal conditions and within certain limits). In addition, dilatation represents a physiologic stimulus for hypertrophy of the muscle fibers (v. Weizsacker, Bohnenkamp) so that with persistence of the working conditions ventricular hypertrophy occurs

Dilatation from increased resistance involves at first almost exclusively the longitudinal dimension of the ventricles, beginning at the end of the outflow tract, progressing toward the apex, and only then involving the inflow tract (Kirch, Jarisch and Loes). In this way characteristic alterations develop in the roentgen image of the heart which differ depending upon whether the right (p 143ff) or the left (p 149ff) ventricle is affected. The understanding of these changes in shape *will be facilitated by reference to figure 65a, b, and c, in which the projections* of the inflow and outflow tracts of both ventricles are represented in the different typical fluoroscopic positions.

Dilatation from increased filling differs from that of increased resistance fundamentally by virtue of the fact that from the beginning not only the longitudinal but also the transverse dimension of the ventricles is affected. This dilatation always prefers that part of the ventricle into which the added quantity of blood flows during diastole, that is, the inflow tract with influx from the atrium, the outflow tract from retrograde filling through incompetent semilunar valves, in aortic insufficiency for example (Zdansky). Naturally dilatation from increased filling is followed by hypertrophy of the ventricular wall. The extent of this dilatation, which gradually progresses over the entire ventricle, depends upon the *amount of blood which flows* into the ventricle in diastole, the filling pressure to which the ventricle is subjected, as well as different constitutional and conditional manners of myocardial reaction Dilatation from increased filling may be too trifling for definite roentgenologic demonstration so that no actual enlargement of the transverse cardiac diameter is *present*, on the other hand it may also become rather considerable without muscular incompetence

With this we are in the midst of the problematic interpretation of dilatation of the ventricles demonstrated roentgenologically. According to the opinion widely prevailing at present, every transverse expansion of a ventricle, roentgenologically demonstrable, indicates muscular failure. Therein one refers primarily to Moritz who called such expansion a "myogenic" dilatation. This view also seems to have received support from the pathologic studies of Kirch on the hearts of athletes, highly trained for endurance, who died from intercurrent disease, and on the hearts of rats who were subjected to a prolonged swimming program Necropsy revealed in man and in animals in all cases, besides hypertrophy of the walls, practically pure elongation but no transverse expansion of the ventricles Even if the ventricles of freshly sacrificed rabbits are filled under high pressure, there is merely longitudinal tension (Monks) From this Kirch concludes that also in living subjects, increased reflux into the heart—just as work against heightened resistance—merely elongates the ventricle and that transverse expansion only occurs with myocardial failure.

At this point it may be remarked that the situation in a dead heart on which these reports are based, need not prevail in living ones which have available diverse possibilities of reflex, vegetative, and hormonally controlled adaptations to altered hemodynamic conditions.

Actually, many observations on living people contradict these demonstrations on dead hearts. Not rarely roentgen examination discloses a universal dilatation of one or both ventricles for which increased diastolic filling comes into consideration as the primary if not exclusive cause while every point suggesting myocardial failure is lacking

This holds, for example, in the cardiac enlargement of many athletes trained for endurance and for long distance running (p 122f).

Dilatation from increased filling, which has nothing to do with myocardial failure, is also involved in persistent ductus arteriosus (p. 440) and in acquired arteriovenous shunts of the systemic circulation Likewise in the dilatation of the hypertrophied left ventricle in mitral regurgitation, increased filling plays a role (p. 180).

It should be expressly emphasized that the dilatation from increased filling has nothing to do with myocardial failure or even a failure of cardiac efficiency, it is, on the contrary, an adequate means used by the intact heart to perform its work under adverse hemodynamic conditions

The same holds true for the longitudinal dilatation of the ventricles frequently found after long standing work against increased resistance. It is true, the normal heart in the living body does not follow the rules of Starling's heart-lung preparation and we may miss every trace of enlargement from increased resistance, it may even become systolically smaller by more complete emptying of the residual blood (Scherf and Zdansky, Reindell, Cabrera and Monroy). But, if this systolic overloading lasts for years, the normal ventricle may dilate in a longitudinal direction (Zdansky). This dilatation is not a sign of myocardial failure and does not mean a failure of cardiac efficiency, it is a satisfactory way for the heart to perform its work under the hemodynamic condition of increased resistance.

Dilatation from increased resistance as well as from increased filling may at any time be converted into dilatation resulting from myocardial weakness when the

ventricle becomes progressively unable to empty properly as the result of a muscle injury or an acute overload. In this type of dilatation which initially represents an attempt to maintain cardiac efficiency, *progressive universal expansion of the ventricle occurs.* When superimposed upon dilatation and hypertrophy from increased resistance, the ventricle is transformed since transverse expansion is added to its elongation (p 144). On the other hand, if it is added to dilatation and hypertrophy from increased filling, the ventricle need not undergo any essential transformation but merely show a simple increase in the universal expansion already present. In general, the appearance of dilatation due to muscular weakness effaces the morphologic differences between dilatation from increased resistance and that from increased filling. This holds particularly for the left ventricle whose shape in muscular weakness, as a rule, no longer permits one to distinguish whether greater peripheral resistance or increased diastolic filling was primarily responsible for the dilatation and hypertrophy In these cases, the diameter of the aorta, demonstrated roentgenologically, alone provides important evidence (Zdansky). The absence of aortic dilatation makes improbable dilatation and weakness of the left ventricle from increased resistance and favors dilatation from increased filling, striking dilatation of the aorta, with proper reservations, favors the opposite conclusion (p 146). In regard to the right ventricle, the roentgenologic demonstration of the absence or presence of a mitral lesion or emphysema provides evidence for the decision whether the dilatation and hypertrophy should be considered the result of increased diastolic filling or heightened resistance (p 143).

In functional respects, dilatation for the preservation of efficiency, while the *result of muscular weakness, still is not an unconditional expression of decompensation*, that is, of failure. At first, dilatation from muscular weakness has compensatory value Although such ventricular dilatation is imposed by defective systolic emptying, by increased amounts of residual blood, still it is an appropriate means for the weakened muscle to maintain efficiency for a while. Fick's principle holds within certain limits, the greater the muscle tension before contraction, the greater the working efficiency of the muscle Actually such hearts can be considered more or less efficient although their reserve power is profoundly reduced

Failure of cardiac efficiency (decompensation) is roentgenologically presumed only when one can prove or infer the signs of retrograde stasis of blood from a ventricle into its atrium, the lungs, or the general circulation In no instance does the degree of roentgenologically demonstrable dilatation of a ventricle permit a conclusion on its actual efficiency. There are greatly dilated ventricles which perform relatively well and only slightly dilated ones with inadequate performance.

Finally, brief allusion should be made to the point that with ventricular dilatation from increased filling, its associated atrium can enlarge without retrograde stasis of blood, that is, without ventricular decompensation We actually found such atrial enlargement—as previously noted—in large acquired arteriovenous shunts of the peripheral circulation, in patent ductus arteriosus, and in some athlete's hearts. Reindell has seen atrial enlargement, at first transiently, after a single physical effort; Kirch saw it in rats subjected to a strenuous swimming program and v. Braunbehrens and Reindell observed it as a permanent event in some athletic hearts Certainly, it involved atrial dilatation from filling under augmented pressure to

which the atrium is subject from these added burdens In other words, the atria now dilate from great filling and not retrograde congestion from the ventricles.

Pure ventricular dilatation resulting from myocardial damage is universal and enlarges the cardiac shadow on all sides when both ventricles are equally affected and no special working conditions exist for one ventricle only (for example, hypertension or emphysema) In accordance with the manner in which the heart fills, this dilatation leads to a balloon-like or bowl-like cardiac shadow perched broadly on the diaphragm. The first form is most frequent in acute myocardial injuries while the peripheral circulation is still relatively intact (paroxysmal tachycardias). The second form is observed in gradually developing cardiac injuries when the circulation is partly affected while the heart is, to some extent, relieved, this is found in myocarditis of varying etiology and frequently in severe anemias.

I. Hypertrophy and Dilatation of the Right Ventricle

One side of the right ventricle rests broadly on the diaphragm and the other forms most of the anterior cardiac wall Only to a limited degree does the ventricle participate in forming the supradiaphragmatic section of the right cardiac border. On the left side its conus comes very close to the border without normally contributing to it

The inflow tract of the right ventricle (fig 65) extends almost transversely from the tricuspid ostium just above the diaphragm and inclines only a little to the left at the cardiac apex. The outflow tract, supported almost perpendicularly by the diaphragm, rises vertically toward the pulmonary ostium to arrive very near the left cardiac border at the conus.

In the anterior view little is seen of the right ventricle. Only with rotation out of the posteroanterior position does it form more of the border. In the left anterior oblique position the ventricle appears below the gradually receding right atrium, until at an angle of rotation of about 40 degrees Then, the ventricular septum runs approximately in the path of the x-rays and the right ventricle forms nearly all of the lower half of the right border (fig 25) In the right anterior oblique position the right ventricle forms a considerable portion of the border. With a rotation of about 60 degrees, at most only a short section of the left ventricle is seen in the supradiaphragmatic section of the left border while the rest is formed by the outflow tract of the right ventricle, at the base the flat bulge of the conus and the initial part of the pulmonary artery project (figs 19 and 65b).

1. *Pure Hypertrophy of the Right Ventricle*

In the anterior view pure hypertrophy of the right ventricle produces at most increased prominence of the pulmonary arc as an expression of a dynamic or fixed dilatation of the pulmonary trunk, in addition the hilar shadows show systolic-expansile pulsations from greater systolic distension of the pulmonary branches (see below). When the ventricle forms much of the border in both oblique positions, increased rounding may be more or less distinct.

Hypertrophy with dilatation of the right ventricle produces diversified altera-

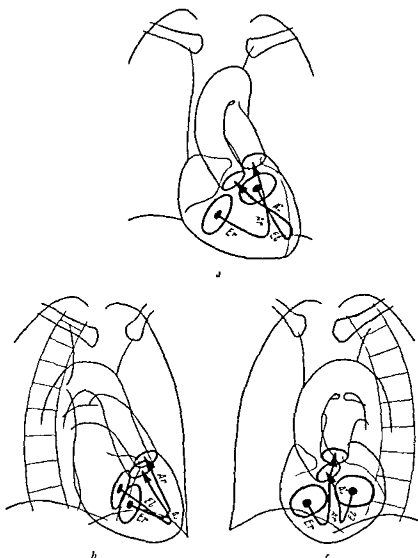


Fig 65 —Course of inflow and outflow tracts of both ventricles in (*a*) anterior view, (*b*) right anterior oblique view, and (*c*) left anterior oblique view Er = inflow tract of right ventricle Ar = outflow tract of right ventricle El = inflow tract of left ventricle Al = outflow tract of left ventricle

a The inflow tract of the right ventricle (Er) inclines only a little toward the apex from its almost horizontal orientation from right to left Therefore, even pure elongation produces widening of the cardiac shadow in the anterior view

The outflow tract of the right ventricle (Ar) passes from the apex almost perpendicularly toward the pulmonary ostium and finds a firm abutment in the diaphragm Therefore, its elongation can proceed only upward to lift the conus and the pulmonary artery higher, this results in

apex Its elongation produces no discernible change in the cardiac shadow in the anterior view. Only with widening in a transverse direction does it expand the cardiac shadow to the left

The outflow tract of the left ventricle (Al) forms the left ventricular arc It extends from the

tions depending upon whether the dilatation results from increased resistance or increased filling.

2 Dilatation and Hypertrophy of the Right Ventricle Due to Increased Resistance

Dilatation resulting from increased resistance which one may find in emphysema, in mitral lesions, or in primary pulmonary sclerosis leads—as was shown on page 138—to almost pure elongation of the ventricle; this begins and is most marked at the end of the outflow tract, gradually advances toward the apex and only then involves the inflow tract. Simple elongation of the outflow tract is able to change cardiac shape characteristically since it fills the cardiac waist and leads to mitral configuration (fig. 66) The outflow tract, rising almost perpendicularly, finds the diaphragm a firm opponent to elongation so that it can develop only upward. Consequently the widened conus pulmonalis and the pulmonary artery are elevated. Since the conus is already located very near the left cardiac border, now it can emerge as a bulge above the left ventricular arc, over it projects the somewhat dilated pulmonary artery This protrusion of the conus and the pulmonary artery is favored by simultaneous rotation of the entire heart around its long axis whereby the right ventricle rotates to the left (Assmann, Kirch) In this way the dilated conus gains a determining influence on the course of the left cardiac border. Since it shifts the left atrium before and gradually flattens it, the conus is able to displace the entire atrium

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apex upward and to the right toward the aortic ostium Lengthening of it elongates the left ventricular arc whereby the cardiac apex moves downward and somewhat to the left on the left diaphragm No noteworthy widening of the cardiac shadow occurs. This happens only when expansion of the outflow tract in a transverse direction is added to the elongation

b In this projection the inflow tract of the right ventricle (Er) appears markedly shortened owing to its practically transverse course Neither its lengthening nor widening in a transverse direction consequently produces any essential change in the right anterior oblique position

The outflow tract of the right ventricle (Ar) forms the border at the left anterior limit of the cardiac shadow Through its elongation there is elevation and a curved protrusion of the conus pulmonalis Moreover, by its transverse expansion, the anterior wall of the heart becomes more prominent and rounded.

The inflow tract of the left ventricle (El), owing to its decided dorsoventral course, together with the size of the left atrium bordering dorsad on the ventricle determines the extent of the cardiac shadow in depth Elongation of the inflow tract is expressed by displacement of the posterior cardiac wall backward, that is, by narrowing of the retrocardiac field from in front This compression must be distinguished from one produced by enlargement of the left atrium (p 165)

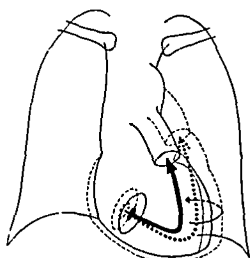
By lengthening, the outflow tract of the left ventricle (Al) produces no essential change of the cardiac shadow in this projection because of its course from the left, below and anterior, to the right, above and posterior, except, at most, deeper extension of the cardiac apex along the left diaphragm

c The inflow tract of the right ventricle (Er) in this projection appears markedly shortened. Its elongation produces no detectable change in the cardiac shadow Only transverse expansion leads to increased bulging of the right cardiac border

By elongation, the outflow tracts of both ventricles (Ar and Al) likewise produce no definite alteration in this projection.

The inflow tract of the left ventricle (El) like that of the right appears greatly shortened in this projection Consequently, its elongation does not change the cardiac shadow essentially Only through its transverse expansion, does the cardiac shadow bulge markedly into the vertebral shadow, while the right cardiac border remains unaffected

backward and to overlap it so that finally the conus becomes part of the border (Assmann) In roentgenograms naturally it is not always certain whether a bulge of this kind developing in this region and forming the border is always the conus pulmonalis or a small wedge of the flattened left atrium projecting over the conus. Below the bulge, the left ventricular arc descends rather abruptly to the diaphragm


FIG 66

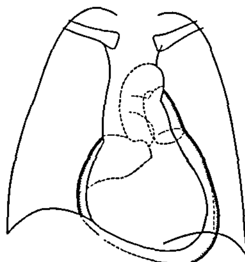

FIG. 67

FIG 66 —Alterations of the anterior view by hypertrophy and elongation of the inflow and outflow tracts of the right ventricle

The elongation of the outflow tract lifts the dilated conus section of the right ventricle and its widened pulmonary artery Through this and through rotation of the entire heart to the left, the cardiac waist is filled. The rotation of the heart results in the sulcus longitudinalis anterior arriving very near the left cardiac border, part of which is formed by the right ventricle. The lengthening *of the inflow tract is expressed by slight widening of the cardiac shadow to the right*

FIG. 67 —Alteration in the anterior view by hypertrophy and dilatation of the right ventricle in a longitudinal and transverse direction The enlarged heart of mitral configuration is widened predominately to the left whereby the left ventricle is displaced increasingly dorsad and vanishes behind the right ventricle Most of the left cardiac border is formed by the right ventricle

The mitral configuration is the immediate result of elongation of the outflow tract of the right ventricle (Német and Schwedel).

As long as the elongation of the right ventricle is limited to the outflow tract, there is no transverse enlargement of cardiac diameter. Only when dilatation includes the inflow tract, corresponding to its almost transverse orientation, does the heart with mitral configuration become broader; often this is predominantly to the right (fig. 66). Therefore the right cardiac arc appears longer, more rounded, and often descends somewhat laterad so that the cardiodiaphragmatic angle is less pointed and the heart is mounted more broadly on the diaphragm Now the right ventricle may form much of the right cardiac border since the right atrium is displaced to the right, backward and upward (Zehbe, Fetzer).

Marked enlargement of the cardiac shadow from dilatation as the result of increased resistance occurs only when dilatation due to myocardial failure supervenes, that is, when the right ventricle dilates from failure; as was explained on

page 139, dilatation from actual incompetence is characterized by universal expansion of the ventricle Then the shadow expands predominantly to the left in accord with clinical and necropsy experience, in other words, in its enlargement the right ventricle makes an attempt to expand toward the left (fig. 67). Consequently, it is improper to infer enlargement of the left ventricle simply from an increase of Ml or of the right ventricle from a larger Mr as often happens Such decisions necessitate a detailed analysis of the cardiac shadow. A left ventricle of normal size is progressively pressed backward by the enlarging right ventricle whose conus section appears more and more along the upper left cardiac border The junction between the left and right ventricles usually is marked by a shallow groove which moves downward with increasing right ventricular enlargement so that the left ventricular arc shortens. The sulcus longitudinalis anterior runs approximately along a perpendicular drawn from the notch to the diaphragm (fig 102). Near the apex, the junction of the two ventricles is not detectable.

With extreme dilatation of the right ventricle, the left ventricle may be displaced backward so completely that finally the entire left border of the heart is formed by the right ventricle

The situation is different when the left ventricle is also enlarged. Then, the enlarged right ventricle finds no place on the left side and must develop more or less to the right Consequently the cardiac shadow expands progressively to the right The right cardiac arcs are lengthened upward and descend somewhat laterad so that the angle formed with the diaphragm is approximately a right angle or is even obtuse Sometimes a shallow notch indicating the atrioventricular junction can be seen (Zehbe).

Examinations in the oblique positions have great importance in decisions about the size and shape of the right ventricle In the right anterior oblique position, elongation and rounding of the outflow tract of the right ventricle, augmented by hypertrophy, as well as the dilatation of the conus and trunk of the pulmonary artery is easily recognized by elevation and protrusion of the pulmonary arc The left anterior oblique position (fig 68) has particular significance for detecting transverse expansion of the right ventricle. Even a simple thickening of its wall is expressed in augmented rounding of the anterior cardiac contour With transverse expansion of the ventricle, the cardiac shadow bulges markedly in a lengthened arc toward the anterior chest wall Occasionally the atrioventricular junction is indicated by a shallow notch in its upper third The left border of the cardiac shadow is not usually essentially altered in its position or course despite marked dilatation of the right ventricle

Frequently the mitral configuration is missed when the diaphragm is low This descent robs the heart of its normal support so that it moves downward and median (p. 175). Under these conditions, if the outflow tract of the right ventricle lengthens, the ventricle develops downward rather than upward since it has lost its firm abutment on the diaphragm Consequently the expanded infundibulum and dilated pulmonary artery are not lifted upward and do not fill the cardiac waist (figs 69 and 93a, b). In other words descent of the diaphragm opposes the mitral configuration. Even when the cardiac waist becomes more shallow owing to the oblique position of the heart and diminished curvature of the left ventricular arc (p 171),

typical mitral configuration is less prone to occur under these positional conditions since the cardiac *rotation to the right, mentioned on page 148, is absent.*

If the dilatation of myocardial failure is added to the dilatation and hypertrophy of the right ventricle which resulted from increased resistance, and the diaphragm is low, the enlarging heart seeks to attain a median position and bulges to right and left with markedly rounded contours. Such hearts can create the impression of aortic configuration resulting from hypertrophy and dilatation of the left ventricle (fig. 158a) The presence of emphysema and of systolic-expansile hilar pulsations naturally warn that one should be careful for they speak in favor of right heart enlargement Moreover, measurement of the aortic diameter provides valuable evidence since a hypertrophied left heart—especially in advanced years—almost invariably is accompanied by aortic dilatation If dilatation of the aorta is absent, in dubious cases this makes it improbable that hypertrophy of the left heart is present and speaks in favor of right heart hypertrophy (fig. 158a).

Belonging to the picture of right ventricular hypertrophy is more or less distinct enlargement of the hilar shadows and increased systolic-expansile pulsations The hilar shadows enlarge owing to high pressure in the powerful hypertrophied right ventricle which produces a dynamic and then fixed dilatation of the pulmonary artery and its intrapulmonic branches This is not pulmonary stasis for the hilar shadows are sharply outlined while in passive congestion they are blurred and demarcation is vague. Schwarz first noted the augmented intrinsic pulsations without interpreting them correctly. Since hypertrophy of the right ventricle greatly increases systolic pressure in the pulmonary artery (Savini), the intrinsic pulsations are *systolic and expansile They are readily distinguished from transmitted pulsations*, the latter are directed purely laterad when the hilus is moved by the adjoining large vessels These intrinsic pulsations are not, however, pathognomonic of right ventricular hypertrophy for they are observed with excited cardiac action in fever and in thyrotoxicosis (Gerhartz) as well as when filling amplitude is large (persistent ductus arteriosus, pulmonary regurgitation, and some septal defects)

3 *Dilatation and Hypertrophy of the Right Ventricle Due to Increased Filling*

Dilatation and hypertrophy of the right ventricle from abnormally increased diastolic filling (p 138) leads to diverse changes of ventricular and cardiac shadow depending upon whether the added blood enters from the atrioventricular ostium or backward through the pulmonary ostium (Zdansky)

With retrograde filling which is possible only with pulmonary regurgitation, dilatation of the ventricle begins and is most marked where the abnormal diastolic influx of blood occurs, that is, at the end of the outflow tract (conus pulmonalis), then it gradually continues toward the apex, finally to involve the inflow tract (p 138) Dilatation from retrograde influx of blood leads to anatomic effects broadly resembling those of dilatation due to increased resistance but the conus pulmonalis tends to dilate more markedly. Pure dilatation of the right ventricle from retrograde filling is rare *and usually congenital (p 285). In the common relative* (Graham-Steell) pulmonary regurgitation (p. 177) dilatation from retrograde filling summates with dilatation due to increased resistance from the coexisting mitral lesion which acts in the same direction.

Dilatation and hypertrophy of the right ventricle from moderate diastolic overloading from the atrium is characterized by elongation, moderate widening, and hypertrophy of the wall which begins and is most marked in the domain of the inflow tract where the augmented blood stream comes through the atrioventricular ostium, gradually to extend toward the apex to involve the outflow tract. Corresponding to its practically transverse course (fig 65a), lengthening of the inflow tract broadens the cardiac shadow whereby both borders extend farther laterally and the right

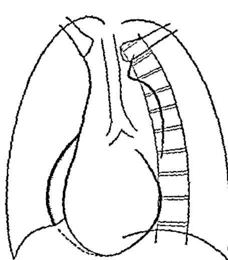


Fig 68

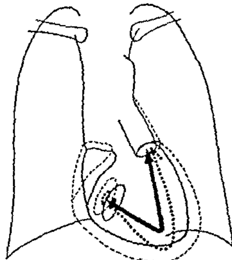


Fig. 69

Fig 68 —Alteration of the left anterior oblique view by transverse widening of the right ventricle. The right border of the cardiac shadow is elongated upward and protrudes with greater roundness into the right lung field

Fig 69.—Dilatation and hypertrophy of the right ventricle with low position of the diaphragm The loss of normal support for the heart results in no elevation of the dilated conus and pulmonary artery despite lengthening of the outflow tract of the right ventricle, consequently, the cardiac waist is not filled Moreover, no rotation to the left occurs The heart seeks a median position and shows a globular shape which closely resembles an aortic configuration.

cardiac arcs also descend laterad In this way the cardiac shadow assumes a noncharacteristic shape with some resemblance to an aortic configuration. The cardiac waist is scarcely filled by widening of the conus and the pulmonary artery in pure dilatation and hypertrophy resulting from increased filling. Moreover, the hilar shadows are still unaltered Furthermore the dilatation is not limited to the ventricle *but always affects the right atrium* as well, since this dilates *under the influence of* increased filling pressure The left anterior oblique position usually shows distinct bulging of the heart toward the anterior chest wall indicating that the enlargement should be referred to the right heart. At all events a normal aortic diameter speaks in favor of dilatation and hypertrophy of the right ventricle

This moderate and rather noncharacteristic dilatation and hypertrophy of the right ventricle due purely to increased filling appears most commonly in athletes trained for endurance performance and long lasting efforts. If, on the other hand, diastolic overloading of the right ventricle from the right atrium exceeds a certain

degree, the picture changes. The dilatation of the right ventricle increases and spreads from the inflow toward the outflow tract. The conus of the pulmonary artery and the pulmonary artery itself become dilated and lifted upward and the heart as a whole rotates to the left. From this results an enlarged and typical mitral configuration of the heart with prominence of the conus and pulmonary artery. The broad intrapulmonary vascular and hilar shadows show systolic expansile pulsations (hilar dance). This picture is found especially with an atrial septal defect (fig. 183)

A similar picture is produced by pulmonary regurgitation which leads to marked prominence of the pulmonary artery and of the conus by elongation and dilatation of the outflow tract of the right ventricle with dilatation of the pulmonary artery. The latter shows marked pulsation like a Corrigan's pulse.

Very complicated situations exist in some congenital anomalies which can lead to alterations of the right ventricle by increased filling and increased resistance, among them are the various forms of abnormal torsion of the bulbus-truncus division. As will be shown more precisely (p 291), enlargement of the right ventricle is often enormous so that the left ventricle is displaced backward and even may vanish behind it. Now, the right ventricle may project far to the left and backward so that there is a close resemblance to an aortic configuration and, indeed in the anterior view (coeur en sabot) as well as in the left anterior oblique position. Reference will be made later to certain differential diagnostic features from congenital cardiac anomalies.

Variations of practical importance occur in the roentgen image of an enlarged right heart when the diaphragm is low (fig. 69). This descent robs the heart of its normal support so that the conus and pulmonary artery are not lifted by elongation of the outflow tract nor is the heart rotated to the right. Consequently, the cardiac waist does not become filled by the widened conus and pulmonary artery The heart, in general sunken and devoid of its normal lower support, assumes a median position with its mass about equally distributed on both sides of the median line Accordingly the heart becomes more or less spherical so that Vaquez and Bordet called it globular

The decision as to whether an enlargement of a heart belongs to the right, the left, or both halves often encounters insurmountable obstacles in these cases Frequently one assumes left ventricular hypertrophy and dilatation and entirely overlooks the changes on the right border The lateral descent of the right border, the large and often accentuated pulsations of the hilar shadows, and the signs of emphysema naturally support the notion that the right heart is enlarged.

II. Hypertrophy and Dilatation of the Left Ventricle

The left ventricle forms almost the entire left border of the heart and part of the diaphragmatic boundary, only parts of its posterior wall near the base contribute to the dorsal cardiac border above the diaphragm. Dorsad and cephalic to the left ventricle is the left atrium which forms most of the posterior cardiac wall although on the left border above the left ventricular arc, the auricular appendix may appear.

The inflow tract of the left ventricle which is folded between the posterior wall and the dorsal surface of the ventricular septum, runs from the mitral ostium, dorsoventrad to the left, forward and below toward the apex, its apical part lies on the diaphragm while the part near the mitral ostium turns toward the posterior mediastinum. The outflow tract, enclosed by the anterior ventricular wall and the anterior part of the interventricular septum proceeds upward from the apex to the right toward the aortic ostium to form most of the left cardiac border (fig. 65a, b, and c).

Consequently, in the anterior view most of the left cardiac border belongs to the outflow tract of the left ventricle, over this lies a short, flat arc of the left auricular appendage. In this projection the inflow tract does not form the border (fig. 65a) but it finally appears with rotation in the left anterior oblique position (fig. 65c). Above it, the left atrium forms the border to a greater extent and, at an angle of about 45 degrees rotation, when the ventricular septum lies approximately in the direction of the ray, the upper half of the posterior limit of the cardiac shadow is formed by the left atrium and the lower half by the left ventricle. In the right anterior oblique position (fig. 65b) the left ventricle vanishes behind the right although the depth diameter of the cardiac shadow in this position, apart from the left atrium, is determined essentially by the length of the left ventricular inflow tract.

1. Pure Hypertrophy of the Left Ventricle

Pure left ventricular hypertrophy causes only slight change in the cardiac shadow. The left ventricular arc in the anterior view is more rounded. Widening of the aorta, usually present in older people, makes the aortic knob more conspicuous. Both cooperate to excavate the cardiac waist more deeply and therefore an aortic configuration is, at least, suggested.

Only when dilatation accompanies hypertrophy does distinct cardiac enlargement and change of shape appear.

2. Dilatation and Hypertrophy of the Left Ventricle Due to Increased Resistance

Dilatation and hypertrophy due to increased resistance such as occurs in hypertension, aortic stenosis, heavy labor, and some athletics, affects, as was explained on p. 138, at first the outflow tract of the left ventricle. Since this forms the left cardiac border, elongation and greater rounding of the left ventricular arc occurs without transverse widening of the cardiac shadow in excess of normal deviations of heart size (fig. 70). In the early stage, this alteration is imperceptible, as a rule, unless there has been an opportunity to follow its evolution. Moreover, the extension of this elongation into the inflow tract creates no further changes in the anterior view. In short, dilatation and hypertrophy of the left ventricle due to increased resistance initially produces relatively minor changes of the cardiac shadow in the anterior view, the waist may deepen when the aortic knob projects farther to the left owing to dynamic or fixed aortic dilatation. Along the elongated, markedly rounded left ventricular arc, pulsations are enlarged and they remain so during repose (Vaquez and Bordet).

The picture is, however, changed very essentially if, through myocardial damage or (rare) acute overstrain, the heart suffers dilatation from myocardial failure and, as a result, the ventricle dilates transversely (p. 140). *In the anterior view* (*fig* 71) cardiac expansion depends upon greater rounding of the left ventricular arc (Rosler, Holzmann) The apex is rounded Only then has the heart assumed a full blown aortic configuration With significant left ventricular enlargement, the cardiac shadow may extend to the left axillary wall

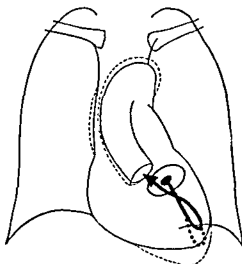


Fig 70 —Alteration of the anterior picture by elongation and hypertrophy of the left ventricle and dilatation of the aorta There is an elongation, directed caudad, and stronger rounding of the left ventricular arc, as well as widening of the vascular band

If the large left ventricle displaces the right heart, the cardiac shadow broadens to the right and this may suggest right heart enlargement Usually this can be excluded by examination in the left anterior oblique position (p. 151).

Since conditions leading to left ventricular hypertrophy are often associated with elevated intra-aortic pressure, as a rule the picture is completed by signs of diffuse expansion and elongation of the aorta (figs. 70 and 71). The resultant radiographic alterations are discussed later At this place it may be said that the resultant widening of the vascular band in the anterior view and the increased protrusion of the aortic arc to the left accentuate the deepening of the cardiac waist already present owing to greater extension of the left ventricular arc as well to the left.

Transverse dilatation of the left ventricle is recognized best in the left anterior oblique position (Vaquez and Bordet, Dietlen) with a rotation to the right of about 45 degrees (fig. 72). Normally, the left border of the cardiac shadow is formed by the basal section of the left ventricle—presuming the level of the diaphragm and the course of the spine is normal—and it descends in a markedly convex arc so that only a small wedge projects into the vertebral shadows and joins the left diaphragm at a sharp angle. This angle lies nearly perpendicular beneath the upper end of the left cardiac border (Zdansky) If, on the contrary, the left ventricle is widened transversely, its more rounded arc projects to the left and posteriorly and extends deep

into the spinal shadows or even beyond them (Arkussky) Accordingly, the cardio-diaphragmatic angle lies rather far to the left below the upper end of the cardiac border. Since this large heart bulges far posteriorly and to the left, and sinks deeply into the diaphragm, the cardiodiaphragmatic angle is blunt and approximates a right angle (Arkussky, Kudisch). On the other hand, the right cardiac border undergoes no noteworthy alteration from left ventricular enlargement; unaltered, it falls

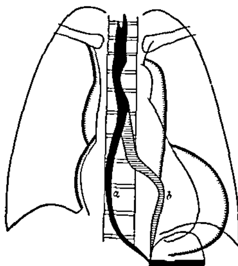


Fig 71

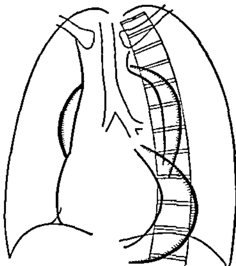


Fig 72

Fig 71 —Alteration of the anterior view by hypertrophy and dilatation of the left ventricle in a longitudinal and transverse direction Owing to the increased bulging and rounding of the left ventricular arc and the cardiac apex, the cardiac shadow assumes an aortic configuration The large left ventricle displaces the right cardiac border so that slight enlargement to the right occurs The vascular band is widened by dilatation of the aorta In the retrocardiac division, the esophagus is either displaced to the right and backward (*a*) or bends into an arc, ventrally convex, to the left (*b*) over the anterior wall of the descending aorta

Fig 72 —Alteration of the left anterior oblique view from hypertrophy and dilatation of the left ventricle in a longitudinal and transverse direction The left cardiac border protrudes far to the left into the vertebral shadow and may even pass beyond it The left cardiodiaphragmatic angle lies to the left and below the upper end of the left border of the cardiac shadow and has become blunt The right cardiac border is scarcely altered The loop of the diffusely dilated aorta *describes a wide arc bulging to the front and back*

vertically to the diaphragm By virtue of this fact, right ventricular enlargement can be excluded with great probability in patients whose cardiac shadow has expanded to the right in the anterior view as the result of right heart displacement.

In the right anterior oblique position (fig. 73), the depth diameter of the cardiac shadow increases (fig. 65b) through lengthening of the inflow tract of the left ventricle In this way the border of the heart facing the posterior mediastinum can encroach upon the retrocardiac field and narrow it since the left atrium is displaced backward. The decision whether the greater depth diameter and the narrowing of the retrocardiac space is produced by elongation of the left ventricular inflow tract or enlargement of the left atrium usually presents no difficulties; in the first instance

the barium filled esophagus describes a broad arc around the posterior cardiac wall (fig. 73) while in the second instance the backward displacement is circumscribed (fig. 84) With marked universal enlargement of the left ventricle the esophagus with its circumscribed displacement sometimes may even be compressed between the large left ventricle and the somewhat dilated descending aorta This displacement, however, involves the supradiaphragmatic section of the esophagus, this is discussed more completely on page 376

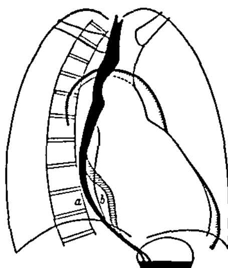


FIG. 73 —Alteration of the right anterior oblique view in a longitudinal and transverse direction from hypertrophy and dilatation of the left ventricle Narrowing of the retrocardiac space with backward displacement of the esophagus in a wide arc (*a*) or in an S-shaped curve (*b*) so that the supradiaphragmatic section moves to the left and forward over the anterior wall of the descending aorta (see fig 71)

In general, pulsations of the left ventricle decrease as the chamber projects farther to the left since, with a fixed stroke volume, pulsations of the ventricular wall must diminish as the ventricle enlarges. The pulsations tend to be smaller in the arc near the apex than in the vicinity of the base (Schwarz) in correspondence with Stumpf's type II pulsations (p. 53). These small apical pulsations may be produced by larger increments of residual blood in this region Thus, the reversal into type I pulsations frequently observed during the exercise test (knee bending, running) may result in the functionally strong heart from stronger cardiac action and more complete emptying of the left ventricle (Reindell).

3. *Dilatation and Hypertrophy of the Left Ventricle Due to Increased Filling*

Dilatation and hypertrophy produced by abnormally increased filling plays a significant role in the left ventricle when there is retrograde diastolic filling in aortic valve regurgitation or increased diastolic inflow from the left atrium in mitral regurgitation, bradycardia, and in certain sports involving endurance and the expenditure of much energy over a long time (see athletic heart).

Retrograde filling of the left ventricle such as occurs in aortic regurgitation leads to roentgenologic alterations of the heart For the same reasons cited in discussing retrograde filling of the right ventricle from the pulmonary artery (p 146), these alterations resemble dilatation due to systolic overload in many respects. In other words, there is dilatation and hypertrophy of the outflow tract, the extent of which depends upon the amount of blood regurgitated; consequently, under some conditions, it may be very slight.

Dilatation and hypertrophy of the left ventricle produced by augmented filling from the atrium predominantly lengthens and widens the inflow tract, this gradually proceeds toward the apex and finally extends to the outflow tract The elongation of the inflow tract is expressed—as was explained above—by lengthening of the depth diameter of the cardiac shadow in the right anterior oblique position while transverse expansion of the ventricle produces greater bulging of the left ventricular arc in the anterior view Usually the apex is plump and round When widening and elongation extends to the ventricular outflow tract, there is increasing elongation of the left ventricular arc which bulges far to the left in the anterior view.

If dilatation from increased resistance or greater filling is superimposed by dilatation as the result of myocardial damage, there is further widening of the heart with its aortic configuration, then, the markedly rounded left ventricular arc may protrude far to the left and almost reach the left thoracic wall. Enlargement of this kind also displaces the right cardiac border more or less to the right

Although such dilatation indicates myocardial damage, this is not synonymous with left ventricular decompensation or failure. This is evident from the fact that patients with this type of heart are often in fairly good physical condition, moreover, roentgenologic signs of retrograde stasis in the left atrium and in the pulmonary circulation are absent (p. 345)

It is even more striking when occasionally these patients have clinical signs of stasis in the greater circulation (enlarged liver, edema, ascites, and so forth) In these relatively uncommon patients the right cardiac border protrudes conspicuously to the right This is the Bernheim syndrome which develops when the ventricular septum is thrust into the lumen of the right ventricle by the enlarged and hypertrophied left ventricle, narrowing it so that inflow stasis develops (Atlas and coworkers, Russek and Zohman, Zdansky)

If this dilatation from myocardial weakness no longer suffices to maintain the necessary working efficiency, then, with a simultaneous increase of size or a noticeable further left ventricular enlargement, retrograde stasis develops in the left atrium and in the pulmonary circulation, in other words, decompensation becomes manifest. Prior to the appearance of a demonstrable enlargement of the left atrium or pulmonary stasis, attacks of cardiac asthma or Cheyne-Stokes respiration may be noted. The cardiac shadow now experiences the alteration designated as mitralization The situations are described in the discussion of the decompensated aortic valve insufficiency on page 187ff.

Radiographic evaluation of left ventricular volume is associated with many sources of error. Thus, elevation of the diaphragm can produce a broadening to the left and an aortic configuration of the cardiac shadow through the transverse position, this may simulate left ventricular hypertrophy and dilatation (p 98). Descent

of the diaphragm can make the left ventricle seem smaller than it actually is owing to median placement of the heart, this prevails in the anterior view as well as in the left anterior oblique position (p. 100) In order to avoid these sources of confusion, it is well to follow Haudek's suggestion correct the abnormal level of the diaphragm by inspiration or expiration Kyphosis of the thoracic spine can obscure enlargement of

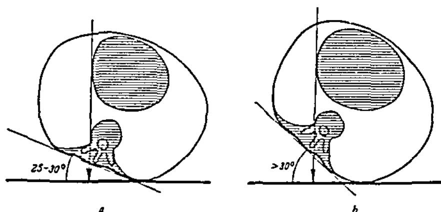


FIG 74 —Determination of the size of the left ventricle by the method of Vaquez and Bordet (*a*) left ventricle of normal size, (*b*) enlarged left ventricle

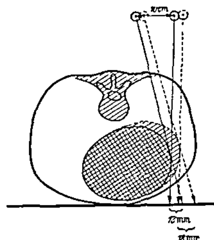


FIG 75 —Determination of the "depth index" of the heart by Vaquez and Bordet Cross hatched left heart of normal size, the parallactic shift of the left border of the cardiac shadow amounts to 12 mm Hatched enlarged left heart, the shift amounts to 18 mm

the left ventricle in the left anterior oblique position since the greater a-p diameter of the chest allows the heart to move away from the spine to widen the retrocardiac space. Finally, in certain cases of cor pulmonale, the right ventricle can develop mainly to the left, displacing the left ventricle backward; whether expansion to the left of these hearts is produced by the left or right ventricle cannot be reliably determined by the criteria previously mentioned Moreover, examination in the left anterior oblique position fails as do the volume determinations recommended by Vaquez and Bordet; nevertheless they will be mentioned for completeness.

In the first, one determines the angle to which the patient must be rotated out of the ventrodorsal direction of the ray (toward the reversed second oblique position) until the left border of the cardiac shadow just touches the left border of the spine (fig. 74) This angle, which, of course, becomes greater when the heart projects farther to the left, normally amounts to 20 to 30 degrees. With left ventricular enlargement, however, it may amount to 40 to 50 degrees. The second method involves the determination of the parallactic shift of the cardiac apex with a target-screen distance of 60 cm. when the tube is moved about 10 cm. to the right (fig. 75). Vaquez and Bordet call this shift the "depth index" of the heart. This progressively increases with greater depth of that part of the heart lying to the left of the spine Normally it amounts to 7 to 14 mm. (average 10) but it may become much larger with left ventricular dilatation As previously stated, this method possesses the same sources of error as simple estimations of volume in the left anterior oblique position The second method has an additional source of error: no consideration is given to the variable heart-film distance which depends upon the shape of the thorax, the position of the heart, or the thickness of the soft tissues over the anterior chest wall (Jalet, Alessandrini)

III. The Right Atrium

To some extent the right atrium curves around the heart from the right, it forms almost the entire right border and, on one side, it extends behind to the left edge of the venae cavae which empty into it and, on the other, forward just ventral to the ascending aorta and just to the right of the conus pulmonalis The sulcus coronarius runs approximately from the cardiodiaphragmatic angle steeply, median, and upward

Accordingly, with sagittal projection, this atrium forms almost the entire right cardiac border, only near the diaphragm does the right ventricle participate, although it is impossible to separate atrium from ventricle (p 20). The right cardiac border projects about the width of a finger beyond the right vertebral border.

With rotation to the left, the ventricle vanishes completely from the right border of the cardiac shadow and the pale shadow of the inferior vena cava now appears between the heart and diaphragm With increasing rotation, cephalad the left atrium imperceptibly replaces the right until, with rotation of about 60 degrees, the left atrium forms most of the right contour of the cardiac shadow (fig. 19a and b)

With rotation to the right, the right ventricle comes progressively into view so that with rotation of 45 degrees the upper half of the right border of the cardiac shadow is formed by the right atrium, the lower half by the right ventricle (fig. 25a and b) If the right atrium fills markedly and becomes rounder during inspiration, often the atrioventricular junction is visible as a shallow notch

Corresponding to its position, enlargement of the right atrium in the anterior view must broaden the cardiac shadow to the right with lengthening and greater rounding of the right cardiac arc Despite these apparently favorable conditions, decisions about right atrial size are not easy. Even the recognition of an occasional isolated and excessive right atrial enlargement (Kronenberger and Leeser, Dressler),

often ascribed to muscular damage of its wall and traction *from without, offers* difficulties In these cases a massive, sharply defined, simply articulated, curved shadow projects into the lung field and may reach from the level of the diaphragm to the bifurcation and from the anterior chest wall to far behind Its *pulsations may* be so small that they are not visible on the screen Consequently, it closely resembles some extracardiac structure broadly in contact with the heart like a cyst, tumor, pericardial diverticulum, or even a mediastinal or pericardial encapsulated effusion

The recognition of right atrial enlargement offers other diagnostic difficulties when—as is usual—other cardiac chambers are also enlarged This depends largely

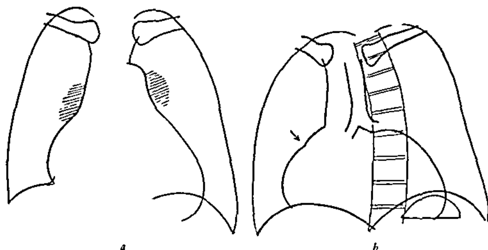


FIG 76—Mitral-aortic lesion with pulmonary congestion in woman, 54 years old The junction between the large hypertrophied right ventricle and right atrium of normal size is evident in the left anterior oblique view (*b*) and is marked by a notch (arrow).

upon the fact that the atrium cannot usually be definitely differentiated from the right ventricle. An expansion of the cardiac shadow to the right need not be due to the right atrium, for enlargement of the right ventricle alone or even of the left ventricle or the left atrium may be responsible The last two possibilities can usually be excluded by analyzing the cardiac shadow, however, a decision as to whether the right heart enlargement involves the atrium, ventricle, or both, very often cannot be made.

As a rule, enlargement of the right atrium is combined with dilatation of the right ventricle. Still, there are cases (tricuspid stenosis for example) with enlargement limited to the atrium, such isolated atrial enlargement may be assumed with some probability if the cardiac shadow, expanded exclusively or predominantly to the right, projects as an elongated rounded arc into the lung field and if, despite the presence of conditions usually associated with heightened pressure in the lesser circuit (coexisting mitral disease), the lung fields are clear and the hilar shadows are sharply defined and not enlarged.

On the other hand, if the right ventricle is markedly dilated, it is highly probable from the start that the right atrium participates in the enlargement. This probability becomes increasingly certain the larger the right ventricle is and the less obvious are

signs of increased pressure in the pulmonary circulation, and when, as a sign of higher venous pressure, the superior vena caval shadow widens. Only when the right heart is enlarged with signs of increased pressure in the pulmonary circuit and the superior vena caval shadow is not widened, should one consider that the enlargement may be confined to the ventricle Sometimes this idea is confirmed in the anterior view when the notch of the atrioventricular junction is high on the upper third of the elongated and laterally descending right heart shadow (Zehbe). Then, in the left anterior oblique position, occasionally a notch (fig 76b) or rounded kink (fig. 77b) is seen just below the ascending aorta on the elongated and markedly



FIG 77 —Mitral-aortic valve lesion with pulmonary congestion in a woman, 53 years old The rounded angular formation (arrow) in the left anterior oblique view (*b*), together with the absence of a widened superior vena caval shadow in the anterior view (*a*) and the presence of marked pulmonary congestion shows that the right heart enlargement is limited to the ventricle

rounded right heart border (Német and Schwedel) To be sure, with greater right ventricular dilatation, these distinguishing marks are often absent despite normal size of the right atrium.

IV. The Left Atrium

The left atrium forms the posterior division of the heart Behind, it borders on the descending aorta and the esophagus, above it, lies the bifurcation of the pulmonary attery and trachea With the diaphragm in normal position the left main bronchus is very near to, or immediately on, the upper limit of the left atrium, but with a low diaphragm, the bronchus is often far beneath this level The pulmonary veins approach the atrium from right and left. On both sides an upper and lower group of these veins widen like funnels toward the atrium The upper venous infundibulum on both sides is ventrad, the lower, dorsocaudad to the main bronchus

The left atrium approaches very close to the right cardiac border since it extends to the left edge of the venae cavae. On the left the auricular appendage regularly forms the cardiac border just above the left ventricle but to an extent individually different (fig 12a and b).

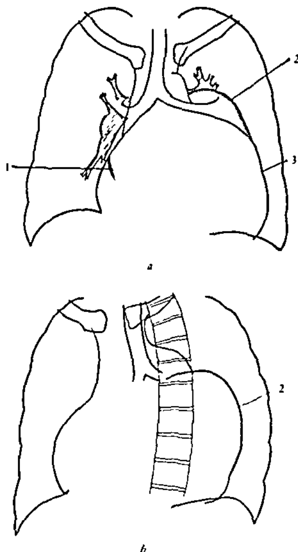


FIG 78 —Mitral-aortic valve lesion with enormous enlargement of the left atrium and of the right heart (*a*) Anterior view The left atrium appears above the gibbus-like projection of the conus of the right ventricle as a systolic-expansile pulsating pale mass and surmounts the left bronchus which proceeds almost horizontally by the breadth of a finger. Moreover, the left atrium is recognized on the right side within the cardiac shadow (*b*) Left anterior oblique view The enormous enlargement of the left atrium is particularly distinct

1 Left atrium
2 Left atrium
3 Junction of conus pulmonalis and left ventricular arc

With a rotation to the left of about 60 degrees (fig 19a and b) the left atrium forms the upper part of the right cardiac border; only near the diaphragm does a small piece of the right atrium still rise over the faint shadow of the inferior vena cava In general this position is optimal for examining the left atrium

With rotation to the right of about 45 degrees (fig. 25a and b) the left atrium forms approximately the upper half of the left border of the cardiac shadow, the

lower half belongs to the left ventricle. Only rarely is the atrioventricular junction visible as a shallow notch.

Corresponding to its dorsal position, an enlarged left atrium compresses the posterior mediastinum and its posterior wall displaces the esophagus. When excessively enlarged, the pulsating left atrium can even erode the vertebra and produce radicular symptoms (Ashworth and Jones).

On the other hand, from its dorsal position a moderately enlarged left atrium need not change shape in any way in the anterior view. Often there is merely a flat bulge in the basal part of the cardiac waist (fig. 90) in which atrial pulsations are

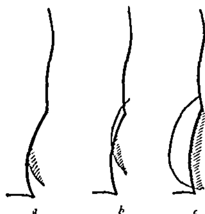


FIG. 79 —Mitral lesion with marked dilatation of the left atrium Female, 23 years old. The left bronchus (arrow) is displaced upward by the enormous left atrium and takes a lateral ascending course. The left atrium together with the upper pulmonary vein *infundibulum developed cephalad in front of the left bronchus* and appears on the left cardiac border as a pale shadow with systolic *expansile pulsations* above the massively enlarged right ventricle which protrudes far to the left

noted With great enlargement a massive bulge may even rise above the left main bronchus and reach the aortic knob (figs 78 and 79). This is possible because the left main bronchus lies behind the upper and in front of the lower venous infundibulum and becomes embedded between them Accordingly with marked enlargement, the ventral part of the atrium with the upper venous funnel can extend upward in front of the left bronchus while the dorsocaudal section bulges below the bifurcation into the posterior mediastinum (Zdansky). In this way the left bronchus is raised and cuts more or less deeply into the atrium between the upper and lower venous infundibula.

Left atrial enlargement becomes obvious on the right side of the cardiac shadow more frequently than on the left. On the right a double contour may form within the right atrial arc or an abnormal arc may project beyond the right cardiac border into

the lung field (Lautemlnicher, Assmann). In the first instance a darker shadow passes median and down in the heart shadow from the middle of the right cardiac border (figs 78, 80a, and 81). In the second instance an abnormal arc protrudes beyond the *right heart border into the lung field*. This arc fills the right cardiovascular angle (fig 80b); it may project beyond the entire right atrial arc and extend down almost to the diaphragm (fig 80c). With very marked dilatation it can directly approach the anterior chest wall to which it may be broadly applied (fig. 100).



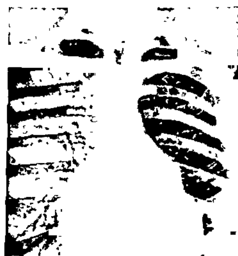


Fig. 80 Fig. 81

Fig 80—Different forms of double contour formation on the right border of the cardiac shadow, produced by enlargement of the left atrium (*a*) The left atrium is visible within the right cardiac border (*b*) The left atrium projects beyond the right cardiac border and even fills the cardiovascular angle (*c*) The left atrium extends far beyond the right and forms the entire right border of the cardiac shadow except for a small supradiaphragmatic section

Fig 81—Mitral stenosis The enlarged left atrium is visible as a dark shadow, convex to the right, within the right cardiac border The bulge protruding above the left ventricular arc belongs to the conus pulmonalis

The appearance of an enlarged left atrium on the right side is greatly promoted by the presence of a mitral lesion since the right ventricular hypertrophy and dilatation rotates the heart to the left (p. 143), this moves the left atrium to the right from behind. In this way, even with slight dilatation, the atrium may appear on the right side as a double contour or a third arc. On the other hand this cardiac rotation *to the left counteracts the appearance of the enlarged left atrium in the cardiac waist*. Actually, in mitral disease the auricular appendix is more or less completely superimposed by the conus pulmonalis which is usually dilated (Assmann).

If the left atrium is relatively large while the rest of the heart is enlarged little or not at all, the atrium can be recognized in thin individuals and children as a dark, oval, nuclear shadow (fig. 82) reaching from the left to the right cardiac border. In

many cases, however, the enlarged left atrium is not visible in the anterior view so that examination in the oblique positions is desirable.

In the left anterior oblique position the upper part of the left cardiac border is formed by the left atrium and the lower by the left ventricle although these chambers ordinarily cannot be accurately differentiated Rather, the left border of the heart usually forms a simple curved arc which cephalad is lost beneath the clear band of the left bronchus in the maze of shadows representing the left pulmonary vessels, only

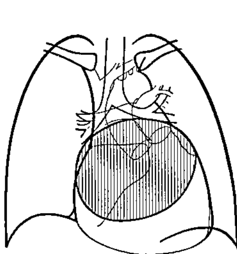


FIG 82

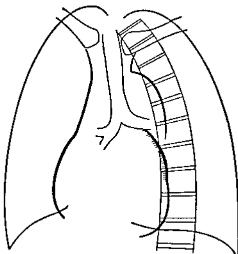


FIG 83

FIG 82 —Projection of the left atrium in mitral stenosis The left atrium forms the borders on the left and right and has spread the bifurcation It may be visible within the cardiac shadow as a darker nuclear shadow

FIG 83 —Mitral stenosis in the left anterior oblique position (schematic) The left border of the heart is elongated cephalad and a subdivision into two arcs may be seen, the upper arc is formed by the enlarged left atrium, the lower arc belongs to the atrophic left ventricle which descends vertically to the diaphragm The left bronchus is displaced upward by the large left atrium and takes an almost horizontal course The right border of the cardiac shadow is also lengthened and markedly rounded, this may be attributed to right ventricular hypertrophy and slight dilatation

in a few cases can the atrioventricular junction be detected as a shallow notch With left atrial enlargement the arc of the left cardiac border is elongated cephalad and is more vertical In place of a simple curved contour there is often a distinct division into a lower ventricular and an upper atrial arc (fig 83), often the latter can be followed to the level of the left bronchus whose clear band may be displaced upward or flattened by the left atrium rising from below, at times the bronchus even proceeds laterad and upward Of course, this dual arc formation is distinct only when the left ventricle is normal or but slightly enlarged, often it is absent with marked left ventricular hypertrophy or dilatation Decisions about the size of the left atrium in this position are further complicated by the fact that the left cardiac border is projected into the spine and into the left hilar shadow which is usually enlarged

Determination of left atrial size is much more reliable in the right anterior oblique position (Assmann) Rotation to the left for about 60 degrees is best In this position,

the right cardiac border, facing the retrocardiac space, has its course determined by the size of the left atrium, normally it descends as a flat arc, convex to the right, vertically to the diaphragm. On the other hand, if the left atrium is enlarged, the right cardiac border bulges decidedly into the retrocardiac field to narrow it more or less (fig. 84). With considerable left *atrial enlargement from the large translucent* area of the retrocardiac space there may remain only a larger clear triangle behind the vascular shadow and a smaller clear triangle above the diaphragm Occasionally the entire retrocardiac space is blotted out.

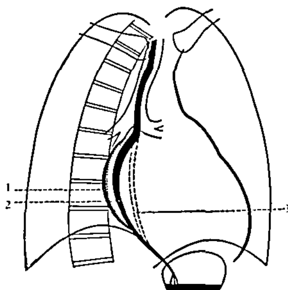


FIG 84—Mitral lesion in right anterior oblique view (schematic) The left atrium bulges far into the retrocardiac field and often a section is projected into the shadow of the spine. There is an angular bend of the barium-filled esophagus below the bifurcation In this particular case it *does not proceed along the right border of the cardiac shadow but is projected* into the left atrium which protrudes posteriorly because it is displaced not only backward but also to the right (The broken line represents the normal course of the esophagus)

1 Left atrium
2 Circumscribed outward bend of esophagus
3 Normal course of esophagus

For reasons cited on page 29 and owing to the pulmonary congestion frequently present in precisely these cases, definition of the posterior cardiac wall and consequently *determination of left atrial size offers great difficulties.* Therefore, ordinarily one cannot avoid filling the esophagus with barium to visualize the posterior cardiac wall indirectly (p. 29) Although originally recommended by Schwarz, this procedure found wide usage after the basic investigations of Assmann and Gabert When the left atrium is enlarged, the esophagus is almost invariably displaced backward.

Stoerck and Kovacs studied the anatomic relations leading to this circumscribed displacement. They showed that the left atrium enlarges below the bifurcation into the posterior mediastium to force its way, so to speak, between both bronchi and

to spread the angle of bifurcation from 70 degrees to over 100 degrees (figs. 82 and 83). Accordingly, the left bronchus, which normally rests on or runs just above the left atrium, can be displaced upward to proceed horizontally or occasionally even pursue a lateral, ascending course (figs. 78 and 79). In children, compression of the left main bronchus with consequent atelectasis of the left lung is not rare (Lauenstein) In adults Zdansky observed stenosis of the left main bronchus and of the right lower lobe bronchus by a large left atrium, provoking disturbances of ventilation and reflex disturbances of pulmonary circulation. These were expressed as emphysema of the involved sections of the lungs and an unusual distribution of pulmonary edema



Fig 85

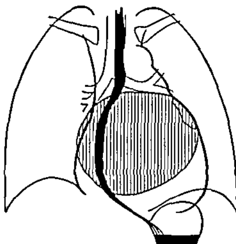


Fig 86

Fig 85 —Circumscribed posterior bend of the barium filled esophagus from enlargement of left atrium (right anterior oblique view)

Fig 86 —Lateral displacement of esophagus by enlarged left atrium (schematic) The barium filled esophagus locally bends to the right (and backward) below the bifurcation The bifurcation is spread by the left atrium pressing from below in such a manner that the left bronchus takes an almost horizontal course (left atrium, hatched)

Elevation of the left main bronchus occasionally leads to paresis of the left recurrent laryngeal nerve

From the close relationship between the esophagus and the posterior cardiac wall (p 29) it follows that increased dorsal protrusion of the enlarged left atrium into the posterior mediastinum cannot help but affect the course of the esophagus At the level of the trachea and at the bifurcation, the esophagus follows its normal course, but, below the bifurcation, it must be displaced, at first dorsad by the enlarged left atrium (figs 84 and 85) The extent of this circumscribed displacement, under otherwise equal conditions, depends upon the size of the left atrium Naturally there is a limit to this dorsal displacement for the esophagus meets insurmountable

resistance from the descending aorta or the spine. Accordingly, in some cases the compression of the esophagus is expressed on transverse projection by distinct narrowing of the lumen and some delay in the passage of the barium paste. Sometimes the latter may be arrested above the narrowing. Nevertheless, disturbances of swallowing are exceptional because the esophagus can also move laterally within the loose connective tissue of the posterior mediastinum

Actually with sagittal projection, below the bifurcation, the barium filled esophagus departs from the midline to bend laterally and, *as a rule, to the right* (fig 86). In a few cases the esophagus escapes pressure from the advancing posterior atrial wall by bending to the left and posteriorly (Rosler and Weiss) This is more common when the left atrium is extremely dilated and extends far to the right (fig 118). Occasionally displacement to the right and left alternate.

Filling the esophagus with barium is the best method for recognizing left atrial enlargement. Naturally no one limits himself to following the course of the esophagus in one position or another A displacement to the left and posteriorly can be missed with rotation exclusively to the right anterior oblique position since with this projection the section of esophagus bulging to the left and backward seems to go straight down Therefore it is necessary to follow the course of the barium filled esophagus in both the posteroanterior and right anterior oblique positions. Naturally displacement of the esophagus is also evident with transverse projections.

In the presence of vasomotor weakness and abnormal cardiac filling from orthostatic factors, left atrial enlargement can be missed with examination in the erect position, it may become demonstrable only with the patient recumbent (Zdansky).

Not every circumscribed dorsal displacement of the esophagus depends upon left atrial enlargement Such displacement may result from hydropericardium (p 324) or elevation of the diaphragm (fig. 22). With the latter, the posterior cardiac wall may press upward and project dorsad so that the esophagus is displaced, just as from left atrial enlargement. On the other hand, descent of the diaphragm may stretch and flatten the posterior cardiac wall so that the left atrium appears smaller than it really is Consequently, consideration of the level of the diaphragm also has great importance in this respect. If the position of the diaphragm is abnormal one should attempt to create a "normal situation for the heart" (Haudek) temporarily by inspiration or expiration in order to minimize resultant errors

Another source of error in interpretation of esophageal displacement arises from *the following fact the position and course of* the *posterior* cardiac wall does not depend exclusively upon the size on the left atrium, the volume of other cardiac chambers exerts an essential influence (p 152) Thus, an enlarged left ventricle displaces the left atrium upward and backward to narrow the posterior mediastinum and the esophagus must bend backward and to the side This bend is, however, distinguished from displacement by an enlarged left atrium by an important feature it is not angular below the bifurcation but usually proceeds as a single large arc over the *posterior cardiac wall (fig 73) This important* differential diagnostic sign is explained as follows· the enlarged left ventricle lifts the base of the heart and displaces the trachea and bifurcation backward and upward without spreading the angle of the bifurcation as happens with left atrial enlargement. Consequently, the esophagus is displaced dorsad even at the height of the trachea and bifurcation, below

the bifurcation, however, there is no longer a circumscribed bend, for the esophagus courses in a long arc over the posterior wall of the large heart

In this connection a circumscribed bend of the esophagus, often to the right, should be mentioned, it lies just below the aortic arch and is produced by the pressure of the left main bronchus (Hall). It is more definitely developed when a dilated pulmonary artery displaces the bifurcation and the left bronchus dorsad against the esophagus (fig. 87). Confusion with an enlarged left atrium is scarcely possible because this outward bend occurs at the level of the bifurcation and ends even above the base of the heart.



FIG 87

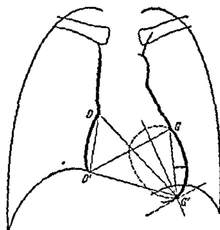


FIG 88

FIG 87 —Circumscribed displacement of the esophagus (arrow) at the level of tracheal bifurcation with dilatation of the pulmonary artery in a case of pulmonary emphysema (Right anterior oblique view)

FIG 88 —Cardiac measurements according to Vaquez and Bordet

These remarks should indicate that the roentgen diagnosis of pathologic alterations of the single cardiac divisions is based primarily on changes of cardiac shape which are naturally associated with changes in its dimensions. These alterations of shape are highly diagnostic but not pathognomonic The interpretations gain importance only by carefully considering the other chest organs, the spatial situation to which the heart must adapt itself, and some pulsatory phenomena. Emphysema, pulmonary congestion or edema, and augmented hilar pulsations are just as important in decisions about the heart as the demonstration of the presence or absence of aortic elongation or dilatation, pulsus celer, pleural effusion, abnormal position of the diaphragm, deformation of the bony thorax (scoliosis, funnel chest), and so forth to which the discussion will return

Vaquez and Bordet attempted to express the size of different cardiac chambers by geometric measurements. They proceeded from four points of orientation (fig

88) Of these points, D lies at the right cardiovascular angle, D' at the right cardio-diaphragmatic angle, G at the lower end of the cardiac waist and G' at a point of the cardiac apex which is found as follows a tangent is drawn on the left cardiac border which runs parallel to a line joining G-D'.

D should indicate the upper limit of the right atrium, D' the junction of the right atrium and ventricle, G the junction of the left atrium and ventricle, and G' the junction of the right and left ventricles near the apex

With the help of these four orienting points, the following measurements are undertaken (fig. 88)

1 GG', the *distance of the left atrioventricular junction from the interventricular junction at* the cardiac apex, this chord of the left ventricular arc measures the length of the left ventricle

2 The longest perpendicular of the left ventricular arc on the chord GG' is the transverse thickness of the left ventricle

3 D'G' the distance of the right atrioventricular junction from the interventricular junction at the apex measures the transverse extent of the right ventricle

4 DD', the distance of the right cardiovascular angle to the right atrioventricular junction, the chord of the right atrial arc measures the size of the right atrium.

5 GD', the distance between the two atrioventricular junctions on each cardiac border, the basal ventricular diameter

6 DG', the distance of the right cardiovascular angle from the interventricular junction at *the apex, the longitudinal diameter of the heart This joins GG' to form the angle DG'G whose* half-length should correspond to the axis of the left ventricle. By mirror-like transference of that part of the cardiac shadow lying on the left of the axis to the right side, Vaquez and Bordet obtain the "contour ovoide" of the left ventricle

The usefulness of these ingenious measurements is sorely hampered by the fact that the location of the four orienting points is associated with important sources of error Irrespective of how easy it may be under normal conditions, it is extremely difficult in pathologic cases and some anomalous positions of the heart Special difficulties are encountered in finding G' which should locate the junction of the ventricles near the apex This point may lie in different places in respect to the roentgenologic apex depending upon the size of the two ventricles If the left ventricle is enlarged, Vaquez and Bordet place it on the border 2 cm inward from the apex, with right ventricular enlarge*ment it becomes the most lateral point of the left cardiac border* Obviously these schematic corrections of the anatomic relations are only approximations The location of the other points also gives rise to many doubts Thus, D' by no means always corresponds to the junction of the right atrium and ventricle since this often lies much higher on the right border (Assmann, Zdansky and Ellinger, Fetzer, and others) Moreover G may lie at different levels even when the size of the left ventricle is constant Its position changes with every cardiac cycle (Cottenot), it moves one or more centimeters upward with diastole According to this author, G *should always be located* in systole There are even more important sources of error If, for example, the heart rotates to the left owing to right ventricular hypertrophy and dilatation, the left ventricle moves posteriorly and is superimposed by the dilated conus pulmonalis so that G lies much lower than corresponds to the left atrioventricular junction Thus, the conception formed of the size of the left ventricle is entirely unreliable It was precisely for the determination of the left ventricular size that Vaquez and Bordet introduced other methods (p 154) and these are also associated with sources of error

To obtain reliable ideas of the size of single cardiac sections, the methods reported by Vaquez and Bordet encounter many difficulties and the values are reliable only in part because the points of orientation often cannot be accurately located Their correct location is possible only by analysis of the cardiac shadow in advance. In other words, this method essentially provides only informa*tion already at hand* The sole contribution of these measurements over detailed analysis of the cardiac shadow is the numeric evaluation of previously discovered alterations For these reasons they have been adversely criticized in recent French literature (Routier and Heim de Balsac)

We intentionally omit numeric definition of the changes of size and shape of single cardiac sections. If this renunciation belabors analysis of the cardiac shadow

with some subjectivity, this is not a crucial objection, exact objective methods of measurements applicable to all cases are still not available. Personal experience is just as indispensible in radiologic analysis of the heart as the equally subjective performance of percussion and auscultation. Overemphasis upon linear measurements endangers a most important fact: the most vital item, precise consideration of cardiac shape, is neglected. The literature contains many examples of misinterpretation which arose from the exclusive employment of measurements, often extremely schematic. This is not intended to deny any value to measurements in roentgenology for every visual method should be supplemented when possible by objective measurements

Chapter Four

The Pathologic Heart

I. The Acquired Valvular Lesions

Valvular lesions are accompanied by abnormal resistances and abnormal diastolic filling so that changes occur in the various divisions of the heart as noted in the preceding chapter

In many respects the situation is, of course, much more complicated One may consider the mechanism of mitral regurgitation. Not only does the left ventricle receive an abnormal amount of blood from the atrium during diastole but part of its content escapes through the incompetent valves in the preceding systole. In this way the work of the left ventricle is very uneconomical, its efficiency is adversely influenced and its content of residual blood increases (Straub) Even without myocardial damage some ventricular dilatation results

In addition to peculiarities of cardiodynamics of this kind, differences of peripheral regulation, of diaphragmatic function, of blood flow through the lungs, or positional relations in the thorax may occur and profoundly affect working conditions of the heart. This should make it clear that gross morphologic changes in cardiac size and shape, roentgenologically demonstrable, can differ vastly from case to case even when aberrations of cardiodynamics seem to be apparently the same. It is precisely these differences that have greatest significance for the evaluation of the individual case because they indicate peculiarities of the myocardium or the participation of certain peripheral mechanisms.

1 *Mitral Stenosis*

Although stenosis of the mitral ostium is associated with regurgitation in most cases, a separate discussion is justified because it is not uncommon for narrowing of the ostium to dominate the anatomic and functional picture completely.

In most cases of mitral stenosis the roentgenogram is very characteristic (fig 89), it is characterized by filling of the cardiac waist, reduction or disappearance of the aortic knob, steep descent of the flat left ventricular arc, and, frequently, visibility of the left atrium on the right side Holzmann speaks frankly of a "mitral stenotic configuration." This is readily explained by the special hemodynamic conditions of this valvular lesion.

A pure or dominant mitral stenosis compels the left atrium to empty against abnormally high resistance. In a minority of cases this resistance can be overcome by hypertrophy without noteworthy dilatation Since it is impossible to recognize

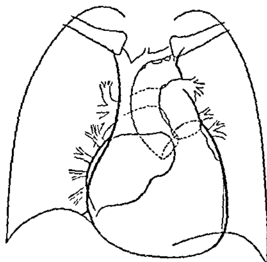


Fig 89 —Schematic representation of mitral stenosis

pure thickening of the atrial wall, in these cases the roentgenogram looks normal. This holds when mitral stenosis has lasted for years as well as in recent cases

As a rule, however, more or less marked atrial dilatation occurs Rarely, this dilatation remains without further dynamic results so that the anterior view shows

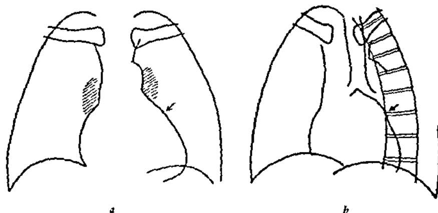


Fig 90 —Mitral stenosis in a woman, 26 years old In the anterior view (*a*) the enlarged left atrium projects as a flat bulge from the cardiac waist In the left anterior oblique position (*b*), enlargement of the left atrium appears distinctly (arrow)

nothing but a bulging in the cardiac waist corresponding to the enlarged auricular appendage, while in the right and left anterior oblique positions, the posterior wall of the heart appears more prominent in the posterior mediastinum (fig. 90). As long as the left atrium overcomes heightened resistance by its hypertrophy, signs of

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The reduction and frequent disappearance of the aortic knob depends not only upon reduced filling of the aorta but primarily on cardiac rotation to the left (Assmann) and the dilatation of the pulmonary trunk just mentioned. The dilated, elongated pulmonary artery rises particularly high to lift the proximal aortic arch enough for the distal section, which normally forms the knob, to vanish behind the pulmonary artery. Consequently measurement of aortic diameter by Kreuzfuchs' method is not possible in these cases (Zdansky). It succeeds only when the aortic knob reappears as the result of elongation and widening of the aorta, this may happen in atheromatosis or combined mitral stenosis and hypertension In the latter the appearance of the aortic knob and hypertrophy of the left ventricle may blot out the mitral configuration of mitral stenosis so that the heart displays essentially an aortic form (fig. 92).

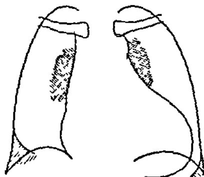


Fig 92—Mitral stenosis and hypertension Woman, 61 years old Systolic blood pressure, 155, two years earlier 185, auricular fibrillation In principle, the heart has an aortic configuration with a shallow cardiac waist, and considerable pulmonary stasis is present The marked dilatation of the left atrium and normal width of the aorta spoke against the presence of a simple, decompensated (mitralized) hypertensive heart and, roentgenologically, also made the existence of mitral disease probable.

The local bulge below the pulmonary arc is produced by the conus pulmonalis, the enlarged left atrium, or both The more the conus dilates and the heart rotates to the left, the more the left atrium disappears behind the conus so that ultimately this alone may form the border (Assmann) The anterior view does not permit one to decide whether the protuberance belongs to the conus or the left atrium unless, as occasionally happens, the bulge vanishes on rotation to the right and projects more on rotation to the left, if this is the case it is formed by the conus, the reverse happens when it corresponds to the enlarged left atrium Naturally, if the atrium projects merely as a small sickle-shaped section over the conus as often happens, the conus and atrium cannot be distinguished

The left ventricular arc is flat and often falls almost perpendicularly to the diaphragm owing to diminished filling and atrophy of the left ventricle (fig 89) Consequently, the transverse cardiac diameter is often strikingly small. Frequently the small size of the left ventricle is first appreciated in the left anterior oblique view as one sees its arc descend vertically to the diaphragm (fig 93a, b)

Only when longitudinal tension of the right ventricle has also extended to the inflow tract does the cardiac shadow slightly widen to the right (fig. 65a) and accord-

increased pressure in the pulmonary circulation or of right ventricular hypertrophy are absent.

For the most part, however, dilatation of the left atrium is associated with increased pressure in the pulmonary veins; the resultant decreased pressure gradient in the lesser circuit loads the right ventricle with increased work which it is able to perform only by dilatation (p. 138) and hypertrophy consequent to higher resistance.

The results visible in anterior views should be clear from the remarks on page 142. Elongation of the outflow tract of the right ventricle with elevation and dilatation of the conus pulmonalis and the pulmonary artery as well as rotation of the heart to the left, leads to more or less complete filling of the cardiac waist, that is, to a



FIG 91.—Mitral stenosis and emphysema Particularly marked gibbus-like protrusion of pulmonary arc.

mitral configuration Increased pressure in the pulmonary artery not only causes dilatation of its trunk but also of its branches and produces enlarged hilar shadows, usually these shadows display intrinsic pulsations in the compensated stage of mitral stenosis. Moreover, the pulmonary arc may also show augmented pulsations.

The bulging and elongation of the pulmonary arc is often striking and, on the average, is greater than one is accustomed to see in other conditions which lead to right ventricular hypertrophy. The marked dilatation of the pulmonary trunk which underlies this situation is, at least, favored by the rheumatic mural damage of the pulmonary artery (Kugel and Epstein, Chiari). Occasionally a spindle-shaped aneursym of the pulmonary artery forms (Spitzer). Marked dilatation of the pulmonary trunk is also found in the common combination of mitral disease and emphysema (fig. 91).

· its transverse diameter enlarges a trifle Then, the right cardiac border is hened and its course downward and lateral

in at least 75 per cent of all cases of mitral stenosis the enlarged left atrium is ble on the right side of the cardiac shadow (Lutembacher, Assmann); this is noted by cardiac rotation to the left (p. 160) Often the left atrium does not ally form the border but appears within the cardiac shadow as a double contour 81, 93) Frequently, however, as a right convex arc it overlaps and fills the t cardiovascular angle (fig. 80b). With further dilatation the left atrium can nd far to the right whereby the right atrium is visible within the shadow of the (fig. 80c) With extreme dilatation the left atrium may form the entire right iac border as a massive curved shadow projecting far into the right hemithorax, nding almost down to the diaphragm, and broadly applied to the right anterior t wall Then, the cardiac shadow is widened considerably to the right The atrium, displaced to the left and forward by the enormous left atrium (Goedel), longer detectable within this dark mass of shadows

The diagnostic difficulties created by these enlargements of the left atrium are light. At first one may consider right atrial enlargement. Against this diagnosis, ever, is the failure of the shadow protruding into the right lung field to extend tly down to the diaphragm, instead, it bends median above it. It is not always ble to exclude absolutely a dermoid cyst, an encapsulated pericardial or medias- effusion or a pericardial diverticulum Most important for the diagnosis is rast-filling of the esophagus a circumscribed deviation below the bifurcation ward and mostly to the left is an almost sure sign for an enlarged atrium.

The stenosis need not be extreme for excessive atrial enlargement to occur edel) In some cases as the cause of such dilatation, a fibrosis of the atrial wall disappearance of the muscle fibers has been postulated, this has interpreted as esult of injury from the previous myocardial disease (Bramwell and Duguid). iese cases it is proper to speak of aneurysmal dilatation of the left atrium embacher)

There is no parallelism between the size of the atrium and the amount of pul- ary passive congestion Precisely when the atrium is especially large, pulmon- stasis may be slight or absent. This situation has been explained by the fact that the right atrium has been narrowed between the left atrium, pressing forward, and the anterior chest wall, to throttle the flow of blood into the heart and therefore into the pulmonary circulation This need not be the only cause, otherwise retrograde congestion in the systemic circulation (liver) would be regularly observed and this is not the case Probably the large left atrium which is capable of holding 2 liters of blood may act as a storage depot to unload the circulation and consequently prevent pulmonary as well as hepatic stasis

On the enlarged left atrium which reaches beyond the right atrium to the right, laterally directed systolic ventricular pulsations are often seen. Luisada and Fleischner actually recorded a plateau-like widening in ventricular systole in electrokymograms. This led them to conclude that there was a systolic reflux of blood from the ventricle into the left atrium and this has anatomic support: stenosis of the mitral ostium is always associated with nonclosure of the valves This reflux may, however, be only slight with high grade stenosis so that it is probable that atrial

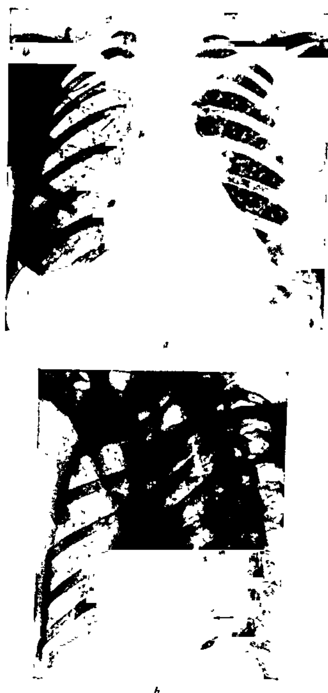
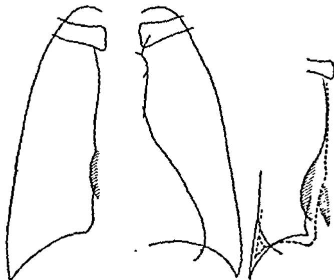


FIG. 93.—Mitral stenosis and descent of the diaphragm. Owing to descent of the diaphragm, filling of the cardiac waist by the dilated conus and pulmonary artery is only slight The left atrium is visible within the right cardiac border as a double contour In the left anterior oblique view the left border of the cardiac shadow is subdivided into an atrial arc and a left ventricular arc which falls vertically to the diaphragm (white arrow). Incidental finding Lob venae azygos (black arrow) (*a*) Anterior view, (*b*) left anterior oblique view.

This rotation to the right favors the appearance of the left atrium on the left side, but it opposes its participation in forming the right heart border, consequently, with diaphragmatic descent, its appearance on the right is rare or less well expressed than with normal diaphragmatic position. Through descent of the diaphragm, the posterior wall of the atrium may be stretched so that the chamber looks smaller than it actually is.



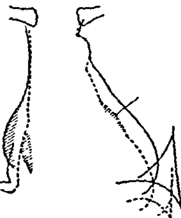


FIG. 94 FIG. 95

FIG. 94 — *Mitral stenosis with marked descent of the diaphragm Male, 58 years old with general paresis Despite widening of the conus and of the pulmonary artery, the cardiac waist is maintained.*

FIG 95 —Reduction of cardiac size and change of its shape in mitral stenosis after a diuresis of eight liters Woman, 46 years old. After the diuresis (dotted lines) the diaphragm descends and the heart becomes smaller The left atrium, previously projecting far to the right, has receded into the cardiac shadow This may depend in part upon reduction of atrial size. This is certainly not the sole reason, for now the left atrium bulges more into the cardiac waist on the left side The change in visibility of the left atrium, at least in part, is the result of rotation of the heart to the right, a consequence of descent of the diaphragm

Elevation of the diaphragm also influences the picture of mitral stenosis. Owing to the transverse position of the heart, this can occasionally oppose the appearance of a mitral configuration (fig. 96).

When cardiac failure occurs in mitral stenosis, different pictures may result

First, cases are not rare in which sudden, repeated but transient pulmonary edema occurs. Occasionally this happens after exertion, delivery, or mental excitement; sometimes rising from bed or the trip to the x-ray laboratory suffices Undoubtedly the pulmonary edema on these occasions is favored by increased amounts of blood advancing from the periphery to the heart and propelled by the hypertrophied right heart into the lungs but no longer disposed of by the left heart (Schellong). This serious catastrophe, frequently fatal, is relatively common with a small or insignificantly enlarged mitral heart with a relatively small left atrium. On roentgen

filling, proceeding briskly under heightened pressure in the pulmonary veins, collaborates in these accentuated pulsations. Moreover, Luisada and Fleischner saw a remarkably small presystolic depression in the atrial kymogram which they considered a sign of very marked stenosis of the mitral ostium or injury of the wall.

When the enlarged left atrium is not visible on the right side of the heart, oblique views, particularly the right anterior oblique, have great importance because the diagnosis of mitral stenosis often depends upon the demonstration of atrial enlargement One may refer to page 161 where the reasons are cited.

Once again, a left atrium of normal size in no way precludes the presence of mitral stenosis There are cases, as was indicated above, in which the atrium overcomes heightened resistance at the ostium by practically pure hypertrophy and thereby compensates for the valve lesion Since atrial hypertrophy is not detectable and since *right ventricular widening and hypertrophy are absent, roentgenologically* it is impossible to recognize these cases which are usually benign and devoid of circulatory disturbances for a long time Ordinarily these patients are women and often without a past history of polyarthritis, tonsillitis, or chorea.

Occasionally, despite normal size of the left atrium there is increased pressure in the lesser circuit and consequently right ventricular hypertrophy which leads to a mitral configuration (Assmann). These cases are rather rare.

In mitral stenosis the left atrium enlarges during the Valsalva test while the left ventricle remains practically unchanged (Pfeiffer). The increased dimensions of the left atrium are explained as follows the excess of blood flowing into the heart with forced respiration cannot pass through the stenotic mitral orifice into the left ventricle with sufficient speed, it stagnates in the atrium By contrast, normally the entire heart decreases in size and left atrial volume diminishes as well Since the latter also happens in mitral regurgitation, Pfeiffer recommends the Valsalva test to aid in differential diagnosis of mitral stenosis.

The most reliable sign for the differential diagnosis between mitral stenosis and regurgitation is the size of the left ventricle if the transverse diameter of the heart shadow is small and if the arc of the left ventricle descends, a little curved and almost perpendicular to the diaphragm, we may assume left ventricular atrophy and the existence and dominance of mitral stenosis, if, on the other hand, the diameter of the cardiac shadow is increased and the arc of the left ventricle elongated, markedly curved, and prominent, it is very probable that the left ventricle is dilated and hypertrophied and that incompetence of the mitral valves prevails. Of course, examination in the left oblique position is necessary for this diagnosis of an enlarged left ventricle (p. 151)

Abnormal spatial conditions in the thorax may influence the picture of mitral stenosis in different ways On page 148, reference was made to the fact that descent of the diaphragm diminishes bulging of the widened conus pulmonalis and pulmonary artery in the cardiac waist and consequently opposes the appearance of a mitral configuration (fig. 94). At the site of the conus the enlarged left atrium frequently appears as a flat arc since the atrium is rotated from the left and posteriorly when the heart turns to the right under the influence of diaphragmatic descent (fig 95). If this flat curved section of left atrium appears above the left ventricular arc, the mitral configuration may be partly preserved.

increase of pulmonary moisture than under normal circulatory conditions (Zdansky). Obviously, then, there is a tendency to greater outpouring of fluids from the vessels, which Eppinger and his coworkers described under the term "serous inflammation." A reliable differentiation between inflammatory exudation and stasis transudation in these cases is impossible roentgenologically and rather improbable at necropsy.

More frequently than augmented flow of blood into the heart, or inflammatory-catarrhal processes in the lungs, increasing cicatricial shrinkage of the mitral valve gradually produces retrograde stasis in the left atrium and pulmonary circuit and consequently signs of increasing pulmonary congestion (p. 345). Occasionally, at this time, the left atrium enlarges Nevertheless, it should be emphasized that the size of the left atrium, the width of the mitral orifice, and the grade of pulmonary congestion are thoroughly independent from each other and precisely when the atrium is largest, signs of pulmonary congestion may be entirely absent while stasis may be most severe in patients with a small or relatively small left atrium

Under otherwise equal conditions, the intensity of pulmonary stasis is proportional to the inability of the left heart to discharge the blood received by it and the amount of blood the powerful right heart pumps into the lungs. Under the influence of high pressure, the arcs of the pulmonary artery and the conus protrude increasingly into the cardiac waist

With failure of the right heart, pulmonary congestion recedes, the hilar shadows become smaller and sharper in outline, the lung fields clearer, and the lung markings less accentuated. This agrees with clinical experience in that dyspnea and orthopnea as well as stasis catarrh and hypostasis in the dependent parts of the lungs diminish with the appearance of right heart failure

Usually, but not always when the right heart fails, progressive dilatation of the right heart becomes increasingly apparent. As noted on page 143 the enlarging right ventricle, providing there is no descent of the diaphragm, tends to develop toward the left unless prevented by an enlarged left ventricle. Since the latter is small in mitral stenosis, the greater volume of the right ventricle expands the heart mainly to the left rather than to the right The right cardiac border bends but little laterad although it becomes distinctly longer and often descends laterally toward the diaphragm; then the cardiodiaphragmatic angle approximates a right angle. Now, most of the left cardiac border is formed by the right ventricle. Whether the enlargement and extension of the heart to the left depends solely on dilatation of the right ventricle or also of the left often cannot be unequivocally settled in the anterior view Fluoroscopy in the left anterior oblique position, however, usually answers this question (p 145).

Graham Steell and Pawinski noted that marked increases of pressure in the pulmonary artery render the pulmonary valves incompetent so that relative pulmonary regurgitation can occur

Scherf discussed the relative frequency of this event, its pathogenesis, and clinical features in detail He observed relative pulmonary regurgitation in mitral stenosis more commonly in females. Frequently, it is transient and often it appears when pulmonary pressure rises and subsides when it falls It unloads the lesser circuit with the diastolic reflux of a certain amount of blood and this explains the disappearance of dyspnea and orthopnea often noted when the regurgitation develops (Scherf) For the appearance of relative pulmonary regurgitation, special flexibility and distensibility of the pulmonary artery is assumed, perhaps this develops on a rheumatic-inflammatory basis



Fig 96 —Mitral stenosis with high position of the diaphragm. The transverse position of the heart opposes the appearance of a mitral configuration



Fig. 97 —Mitral stenosis with pulmonary edema in a febrile catarrhal illness. This male, 31 years old, had mitral stenosis since childhood He was ill for two days with dyspnea, dry cough, and fever On the following night red, frothy sputum was expectorated The remittent fever fell by lysis during the next fourteen days During this time the patchy shadows in the lungs vanished. The cardiac shadow remained unaltered

[illegible] the size and shape of these hearts may not be altered, only the signs of

[illegible] with fever and clinical signs of a catarrhal pulmonary disease (fig 97). We have the impression that such catarrhal processes in mitral disease are often accompanied by a more definite

flattening of the pulmonary arc, and recession of all signs of pulmonary congestion, in other words, *with failure of the right heart pressure falls in the lesser circuit* and the pulmonary artery becomes narrower. Often, however, the anterior view of the heart does not change even when cardiac size changes as demonstrated by percussion. This occasional superiority of percussion is explained by greater or lesser application of the right heart, varying in extent, on the anterior chest wall, this can be *easily demonstrated by percussion* (Wenckebach). Roentgenologically such changes have great significance in cardiodynamics despite their sparseness and are often demonstrable only in oblique or transverse positions, difficulties of an exact reproduction of the position and poor radiolucency of these chests owing to lung stasis often stand in the way.

Pictures similar to those of a relative pulmonary regurgitation occur in Lutembacher's syndrome and in mitral stenosis combined with pulmonary regurgitation *following endocarditis. This is, however, a rather rare combination* (Schwartz and Shelling).

Enlargement of the right heart usually involves the atrium as well as the ventricle. The possibility and difficulty of deciding the extent of ventricular and atrial involvement was discussed on page 156. Fairly marked enlargement of the right heart always speaks with some degree of probability for involvement of both sections. If signs of pulmonary congestion are lacking or are only slightly developed, *if the shadow of the superior vena cava has expanded, or if the right diaphragm is* elevated or a hydrothorax is demonstrable, the presence of a relative tricuspid regurgitation must be considered. Holzmann suggested designating this alteration of the cardiac shadow as the "tricuspid configuration." Lateral pulsations of the right cardiac border in ventricular systole, described by Groedel, were not observed by Assmann, Dietlen, Holzmann, nor Zdansky, roentgenkymographic examinations which could provide an exact answer on this feature apparently are yet to be reported. *In tricuspid regurgitation, Holzmann observed a systolic lateral movement* of the shadow of the left innominate vein, projecting convexly to the left, above the aortic knob (fig. 114).

With restoration of compensation, frequently cardiac size distinctly diminishes as the right ventricle becomes smaller. Often, all signs of pulmonary stasis recur with improved performance of the right ventricle.

The reduction of the cardiac shadow after compensation is restored or after copious diuresis naturally could depend partly or exclusively upon the absorption of pericardial transudate. At present, the opinion generally prevails that this has nearly always happened *when the cardiac shadow becomes smaller in valvular disease.* In this respect one probably goes too far. In many cases analysis of the cardiac shadow before and after compensation shows actual reduction of cardiac size and particularly of those parts which have been subjected to greatest demands. Thus, in mitral stenosis the reduction is often limited exclusively to the right heart. One may assume, a priori, that when the cardiac shadow enlarges during the stage of decompensation a pericardial transudate often participates. To what extent the reduction of the cardiac shadow depends upon diminution of heart size and how much it involves absorption of a hydropericardium, naturally cannot be exactly demonstrated.

and leads to a "dissociation of the valves" (Chiari) Dilatation of the conus pulmonalis favors its occurrence

The roentgen picture of typical relative pulmonary regurgitation is very characteristic (fig. 98). Besides other signs of a mitral lesion, the conspicuous, elongated pulmonary arc *protrudes in a striking manner* from the cardiac waist and below joins the usually flat curve of the conus pulmonalis or left atrium, finally to merge with the left ventricular arc which descends relatively steeply The hilar shadows are enlarged and lung markings accentuated, *nevertheless no severe pulmonary stasis* appears because the regurgitation, as already mentioned, tends to unload the lesser circuit (Scherf) Frequently, but not invariably, the pulmonary arc and hilar

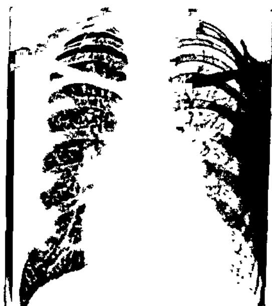


FIG. 98 —Relative pulmonary regurgitation with mitral stenosis

shadows show increased systolic expansile pulsations but they lack the character of pulsus celer save in a minority of cases (Pezzi) The absence of a pulsation like that of pulsus celer, despite the valvular incompetence (Assmann, Holzmann), is considered the result of a small stroke volume in mitral stenosis, this fails to provide sufficient blood pressure amplitude

The roentgen picture is very characteristic It permits one to decide in mitral disease, *when doubt exists, whether a diastolic murmur of regurgitation* audible on the left sternal border is produced by a mitral lesion in combination with an aortic or with a pulmonary regurgitation with increased assurance for the latter conception (Schwartz) Nevertheless, this roentgen picture is not absolutely conclusive for relative pulmonary regurgitation, rather, it merely shows that an anatomic basis for its appearance is present As Scherf stresses, a change in the strength of the heart in these patients is not rare and with it the diastolic murmur of regurgitation disappears and reappears. If this reciprocal play is *followed roentgenologically, in* some cases a corresponding alteration of cardiac size and shape is found, when the murmur of regurgitation disappears, one sees increasing dilatation of the right heart,



Fig 100 —Mitral lesion with massive enlargement of the left atrium which forms the entire right heart border with the exception of a supradiaphragmatic section and which is broadly applied to the right anterior chest wall. Pulmonary congestion is only moderate

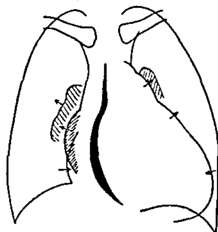


Fig 101 —Alternating pulsations of the left atrium which forms the border on the right and on the right atrial arc in a patient with mitral regurgitation and stenosis The large systolic-expansile pulsations of the left atrium are transmitted to the right hilus (The arrows indicate the direction of the pulsations in single areas)

Usually the left atrium is enlarged, at least in two thirds of the cases it is visible within the right cardiac border or projects beyond the right atrium Often it becomes enormous (fig 100). A left atrium of normal size and exclusively hypertrophied does not occur in mitral regurgitation with an enlarged left ventricle If the left atrium projects to the right beyond the right atrium its pulsations are often striking in size and laterally directed during ventricular systole and they alternate with those of the right atrial arc (Mahaim) (fig. 101) According to Rosler and

2 Mitral Regurgitation

Mitral regurgitation due to rheumatic fever. Pure mitral regurgitation is also uncommon, usually it is combined with stenosis. If, in the following pages, we speak about mitral regurgitation it merely means that the incompetence of the mitral valves stands in the foreground of cardiac and circulatory dynamics.

As in mitral stenosis, normal roentgen findings do not positively exclude the existence of mitral insufficiency, sometimes physical examination is superior Often weeks and months must elapse before a mitral regurgitation resulting from rheumatic fever, clinically established, produces radiographic changes These patients are, however, less common than those in which a systolic murmur, a loud first sound

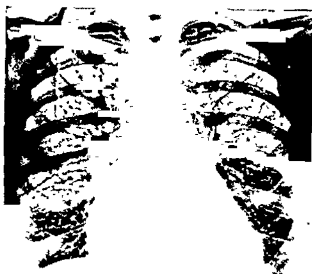


FIG 99 —Compensated mitral regurgitation The moderately enlarged heart of mitral configuration shows elongation and marked rounding of the left ventricular arc and bulges to the right with a lengthened, markedly rounded, and laterally descending arc

at the apex, and a low grade temperature, which could be produced either by a mitral valvulitis or hyperthyroidism, are shown on roentgen examination to be due to a valvular lesion. In silent mitral regurgitation all murmurs are absent while x-ray often permits an unequivocal diagnosis. This emphasizes once again that neither clinical nor x-ray examination is entirely sufficient, they supplement each other.

Roentgenologically mitral regurgitation differs from stenosis by the presence of dilatation and hypertrophy of the left ventricle, that is, by expansion of the cardiac shadow to the left with lengthening and greater rounding of the left ventricular arc and of the apex (fig. 99) (p. 153) This transverse enlargement of the left ventricle should not be considered a sign of myocardial damage. Rather it is the result of the diastolic overloading by the pendulum blood. The second reason is the loss of tension by the regurgitation of blood into the atrium, in this way the aortic valves open too late and the ventricle retains an abnormal amount of residual blood (Straub).

conus and the pulmonary artery (fig. 102). The left anterior oblique position affords good conception of the size of both ventricles (p 145).

Earlier, it was noted that right heart failure in mitral stenosis unloads the lesser circuit and consequently reduces all signs of pulmonary stasis. This also holds for mitral regurgitation, therefore, as a rule, as the cardiac shadow widens to the right, simultaneously the hilar shadows shrink, the lung fields clear, and lung markings become less accentuated. Occasionally these signs of right-sided failure appear without noticeable enlargement of the right heart.

In regard to the diagnosis of relative tricuspid and pulmonary regurgitation (fig. 103) the statements made in the discussion of mitral stenosis hold.

With restoration of compensation the cardiac shadow often becomes remarkably smaller. This does not depend upon absorption of a pericardial effusion exclusively

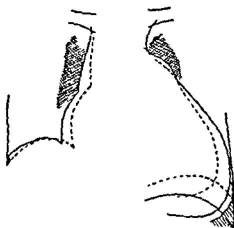


FIG. 103.—Mitral regurgitation with relative pulmonary and tricuspid regurgitation Female, 46 years old (necropsy) Very large heart expanded to left and right, mitral configuration with [illegible]

but, at least in part, upon a major reduction of actual cardiac size. Exact analysis often reveals unequivocally that the reduction of volume does not affect all parts of the heart equally, the parts subject to the greatest demands and most dilated, likewise show the biggest reductions (Zdansky).

Relative or secondary mitral regurgitation Fundamentally the roentgenogram of regurgitation of this type, is determined by the ground upon which it develops The left ventricle is always dilated and widens the mitral ring to render the valves incompetent

Primarily, decompensated mitralized aortic hearts come under consideration, irrespective of whether an aortic valve lesion or hypertensive hearts of various types are involved The roentgenogram of a mitralized aortic heart will be discussed later (p 187b). By anticipation it may be mentioned that in comparison with mitral regurgitation resulting from rheumatic fever, the left atrium usually remains within

Dressler, Lemke, and Luisada and Fleischner, these pulsations of the left atrial arc express sudden atrial distention by blood regurgitating from the left ventricle during systole However, blood entering under heightened pressure from the pulmonary veins contributes to sudden widening of the atrium since similar pulsations are also seen in practically pure mitral stenosis (p. 173).

Undoubtedly the pulsations described by Bedford and Dressler as occasionally palpable between the sternum and right midclavicular line in mitral regurgitation with a large left atrium must be assigned to a left atrium extending far to the right as shown by x-ray.

The left atrium extending far to the right shifts the hilus on this side to the right and transmits to it laterally directed systolic pulsations (Rosler) (fig. 101).



Fig. 102 —Mitral regurgitation and stenosis The broken line drawn caudad from the conus pulmonalis indicates the approximate course of the sulcus longitudinalis anterior

As might be anticipated, the other alterations correspond thoroughly with those enumerated for mitral stenosis. Moreover, the changes of the cardiac shadow in the stage of decompensation are quite analogous to those of mitral stenosis except that left ventricular dilatation is sometimes very conspicuous

As in mitral stenosis, progressive impairment of the discharge of blood from the lesser circuit *also leads to pulmonary congestion which is more strongly* expressed whenever the right heart pumps blood into the lungs with great force With high pressure, the pulmonary artery may progressively dilate and, accordingly, it may protrude farther.

With failure and increasing dilatation of the right ventricle, the heart expands *to the right* since this ventricle must enlarge mainly in that direction when the left ventricle is enlarged. The anterior limit of both ventricles can be determined with good approximation by drawing a line slightly convex to the left caudad from the

border in the anteroposterior position is, in these cases, only the result of its displacement to the right by the large left ventricle

Although one may assume that this kind of left ventricular enlargement reflects myocardial failure, signs of decompensation—passive congestion for example—may not appear for a long time The left atrium also is not enlarged, the lung fields are clear, and the hilar shadows normal These patients may continue their activities and even perform major physical feats.

On the left ventricular arc, the strikingly large, powerful pulsations correspond to the augmented ventricular stroke volume. These pulsations may also be transmitted to the right cardiac border so that both borders show equally large pulsations to the unaided eye Kymograms of the left ventricular arc reveal that the systolic

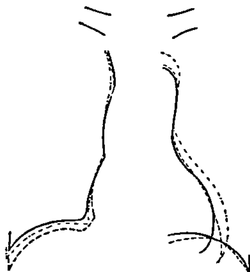


FIG 105 —Syphilitic aortic valve regurgitation and aortitis Increasing elongation and finally transverse expansion of the left ventricle ______February 23, 1933 arch diameter = 2 9 cm, ‐ ‐ ‐ ‐ May 10, 1933 arch diameter = 2 8 cm, - - - June 1, 1934 arch diameter = 3.3 cm

median movement is shortened by the premature onset of the diastolic lateral movement (Stumpf) The pulsations are especially large and flapping when an active endo- or myocarditis exists

The vascular band may be normal in breadth but is usually widened since the aorta is more or less dilated and elongated. Accordingly, the right upper arc of the mediastinal shadow is more conspicuous and lengthened at the cost of the right atrial arc, on the left, the aortic knob projects farther to deepen the cardiac waist and to accentuate the aortic configuration The descending aorta is exposed broadly as a pale shadow in the cardiac waist and can be followed down to the diaphragm within the cardiac shadow

On both borders of the vascular band, often with especial distinctness on the aortic knob, the roentgenologic correlate of a peripheral pulsus celer is seen as pulsations of striking size and characterized by a brisk systolic outward and a slower diastolic inward movement In febrile diseases, particularly in active endocarditis,

modest limits so that it projects less into the posterior mediastinum and only rarely beyond the right cardiac border. Moreover, signs of extreme hypertrophy of the right ventricle and marked dilatation of the pulmonary artery are missing.

3. *Aortic Valve Insufficiency*

Blood regurgitating through incompetent aortic valves causes dilatation of the left ventricle (p. 152) as the result of retrograde filling which at first expands and elongates the outflow tract of the left ventricle. Consequently, no noteworthy transverse enlargement of the cardiac shadow need occur; rather, this dilatation may be expressed merely by lengthening and greater rounding of the left ventricular arc and, at the beginning, this need not essentially alter cardiac shape. Actually, it

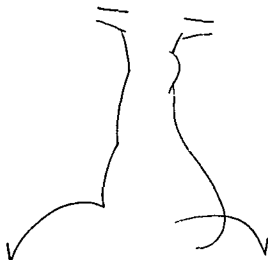


FIG. 104.—Aortic valve regurgitation due to rheumatic fever. The heart is normal in size and the left ventricular arc is somewhat elongated. Pulsus celer on the aorta. This male, 38 years old, was a mountain climber and a former football player. He had rheumatic fever eighteen years earlier.

is not unusual for aortic regurgitation to last for some time without any gross change of cardiac shape (fig. 104).

In most cases, however, the left ventricular arc is distinctly lengthened, more markedly rounded with the apex displaced downward (p. 153) and shows striking pulsations which depend upon the augmented ventricular stroke volume. As long as dilatation from retrograde filling is confined to the outflow tract of the ventricle, one finds no change in the oblique positions.

When the valve lesion has existed for a longer time or the myocardium is damaged, usually the ventricle progressively widens transversely from myogenic dilatation (p. 139). Consequently the cardiac shadow expands to the left, the left ventricular arc and the apex become rounder, and the cardiac waist deeper (fig. 105). Then, the left cardiac border can reach almost to the left axillary wall. Moreover, the mid-right distance can increase somewhat but this should not be casually dismissed as an expression of right heart enlargement. The left anterior oblique position reveals that the right heart is not enlarged. The outward bulge of the right cardiac

sionally they constitute the sole indication of aortic valve regurgitation in a very large mitralized aortic heart.

At first the lung fields show no congestion although these patients often complain of exertional or nocturnal dyspnea or suffer from Cheyne-Stokes respiration. Sooner or later, however, retrograde stasis of blood occurs without the left ventricle necessarily having reached considerable size.

Even before pulmonary congestion becomes demonstrable occasionally the left atrium enlarges It is relatively uncommon for this to occur in the form of double

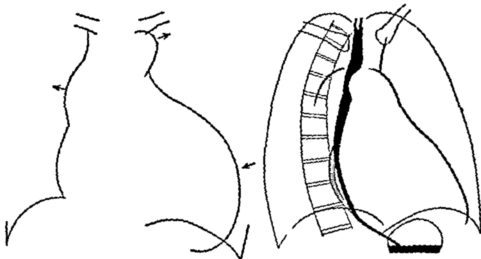


Fig 106 —Aortic valve regurgitation due to rheumatic fever with massive dilatation of both ventricles, the result of myocarditis. Dilatation of ascending aorta Male, 22 years old (necropsy). Very large, lively pulsations of the left ventricular arc and huge pulsus celer on the aorta as often seen in active endomyocarditis. Isolated dilatation of ascending aorta whose circumference was 11 5 cm (The arrows indicate the direction of the large systolic pulsations.)

Fig. 107 —"Mitralization" in failure of hypertrophied and dilated left ventricle in right anterior oblique position (schematic). Retrograde stasis from left ventricle into atrium is recognized by the circumscribed arc of the esophagus (hatched) which is, to some extent, superimposed upon the large bend of the esophagus over the posterior cardiac wall. The circumscribed arc indicates enlargement of the left atrium

contour along the right heart border It is easy, however, to demonstrate atrial enlargement in the right anterior oblique position since the barium filled esophagus locally bends outward, backward, and often to the right above a wide arc which sweeps over the posterior aspect of the large heart (fig 107).

Usually the cardiac waist soon becomes shallow and signs of pulmonary congestion manifest. Then, it is proper to speak of "mitralization" (Vaquez and Bordet) because the cardiac and circulatory dynamics as well as the effect on roentgeno-

[illegible]

ary circuit In this way right ventricular work increases and dilatation from in-

these rapid pulsations are especially large and actually flapping; excursions of the aortic border, 4 to 5 mm. in width, are not unusual. Extremely wide excursions indicate a reduction of mural tension (Zdansky).

Some cases of aortic valve regurgitation do not show a pulsus celer in the aorta. This makes the roentgenologic discovery of the valvular lesion and distinction from hypertensive disease impossible. It seems plausible to explain the absence of pulsus celer by a loss of aortic elasticity from atheromatosis or syphilis. Actually, this is true in some cases. However, at times the rapid pulsations are missed when necropsy discloses trifling changes or a normal aortic wall. This suggests that functional factors account for the absence of pulsus celer, perhaps a heightened tonus of the vessel wall. On the other hand, occasionally an unequivocal pulsus celer is seen despite advanced aortic alterations. Consequently no definite decision about the structural status of the aorta should be drawn from the presence or absence of pulsus celer (Zdansky) The situation merits precise study.

The aortic diameter is important in decisions about the valvular lesion, for the aortic lumen is ordinarily normal or less dilated with regurgitation due to rheumatic fever than in lues. Nevertheless, aortic dilatation can be absent even in syphilis until an advanced age. In our experience this happens mostly in females In some cases, perhaps the aorta was originally narrow so that the diameter does not exceed normal despite mural syphilis

Moreover, long lasting aortic valve regurgitation from rheumatic fever with coexisting hypertension or at advanced ages usually causes more or less marked aortic dilatation so that conclusions about the etiology of the lesion from aortic diameter are unreliable. The difficulty is increased by the fact that in both rheumatic aortic valve regurgitation and in lues the dilatation affects primarily the ascending aorta (Dietlen), the same holds in the active stage of endocarditis and even in young subjects. Often the dilatation mentioned last is dynamic rather than anatomic (Allbutt, Purks), it is an abnormal distention of the aortic lumen resulting from diminished mural tension (tonus) At necropsy of these patients the aorta is normal in width and often rather thin If the active process subsides, the dilatation may vanish On the other hand, it may become anatomically fixed. While the entire thoracic aorta may be affected involvement often occurs predominantly or exclusively in the initial section of the ascending aorta so that syphilitic aortitis or mycotic aneurysm seems likely (fig 106). An aorta, dynamically dilated, ordinarily shows remarkably large, flapping pulsations as mentioned above, these, in conjunction with similar pulsations of the cardiac shadow (p. 212) offer valuable evidence for the existence of an active endo-myocarditis (Zdansky).

With failure, usually the left ventricle dilates progressively, consequently the heart widens farther to the left and the left ventricular arc, projecting strongly but [illegible]

the stroke actually diminishes and partly because the same stroke volume produces smaller excursions on a markedly dilated ventricle than on a smaller one.

Moreover, the pulsations of the vascular band like pulsus celer become less distinct and finally vanish Often, however, they persist for a long time and occa-

Moreover, the conus and the pulmonary artery do not protrude sharply into the waist so that it merely becomes shallow. The difference, so important in differential diagnosis, in respect to the pathologic lesion in the mitral valve may be explained as follows: failure of the left heart raises resistance in the pulmonary circuit more or less acutely without producing as much right ventricular hypertrophy and dilatation of the conus and pulmonary artery as in mitral disease; in the latter, resistance in the pulmonary circulation *develops gradually and causes marked dilatation and* hypertrophy of the right ventricle and marked ectasia of the pulmonary artery. If, for some reason, like a coexisting emphysema, pressure increases in the pulmonary artery and the right ventricle hypertrophies, even a pure aortic valve incompetence may be associated with a dilated conus and pulmonary artery which fills or projects markedly into the cardiac waist. Consequently, in an aortic valve insufficiency combined with emphysema, one is justified in ascribing more or less marked filling of the cardiac waist to left heart failure only when the left atrium is enlarged and pulmonary congestion is present

Obviously, a mitralized aortic valve regurgitation closely resembles combined mitral-aortic disease The possibility of a distinction will be mentioned later (p 198).

Usually mitralization and pulmonary congestion are combined although the amount of pulmonary passive congestion varies greatly. It is true, *ceteris paribus*, that pulmonary congestion is more extreme when the left heart works poorly and the right heart pumps blood powerfully into the lungs When the right heart begins to fail, pulmonary congestion more or less recedes, the lung fields become clearer, the hilar shadows smaller and sharper in contour and the lung markings diminish. Often, simultaneously the heart progressively widens to the right owing to dilata*tion of the right heart (fig 110) Examination in the left anterior oblique position* usually shows that both halves participate in the enormous cardiac enlargement (fig 111) Stasis in front of the heart is often expressed by widening of the superior vena cava and elevation of the right diaphragm (stasis in the liver). In these cases it is possible, indeed probable, that relative tricuspid regurgitation is present

In aortic valve regurgitation after rheumatic fever, both ventricles may dilate enormously owing to myocarditis without the left atrium participating Since both ventricles become equally incompetent, pressure in the lesser circuit may not increase and consequently no pulmonary congestion appears Then, the globular heart is enormous and bulges to the left and right (fig 106), despite its striking size, it may show large, lively pulsations Often an aortic pulse, like that of pulsus celer, is developed very well

Sometimes, despite retrograde stasis in the systemic circulation, right heart enlargement is absent At necropsy, these patients have, on the whole, little or no noteworthy right heart enlargement despite its failure. Occasionally such retrograde stasis is not provoked by right heart failure but by the dilated and hypertrophied left ventricle pressing against the right, narrowing its lumen and preventing the inflow of blood into the right heart Despite the clinical signs of retrograde stasis in the systemic circulation, both pulmonary stasis and right heart dilatation are absent (Bernheim's syndrome)

With a mitralized aortic heart in particular, there is always the possibility that a

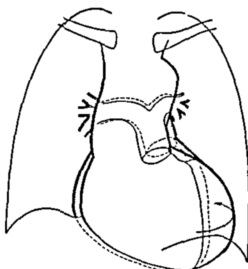


Fig 108 —So-called "mitralization" in failure of hypertrophied and dilated left ventricle (schematic). The dilatation of the right ventricle causes widening to the right and rotation to the left (arrow) as well as elevation of the dilated conus and pulmonary artery As a result the cardiac waist becomes shallow

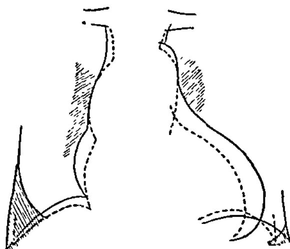


Fig 109 —Luetic aortic valve regurgitation Syphilitic aortitis Female, 60 years old Typical picture of "mitralized" aortic heart with shallow cardiac waist, signs of pulmonary congestion and bilateral hydrothorax. Thirteen months earlier (orthodiagram in broken line) no signs of left heart failure although at that time the left ventricle was already enlarged considerably The ascending aorta is dilated decidedly

creased resistance develops, at first, there is predominant elongation of the outflow tract and later also of the inflow tract with rotation of the entire heart to the left The cardiac waist, previously deeply excavated, now becomes shallow and the lung fields, formerly normal, show progressive hilar enlargement, accentuated markings, and more or less diffuse clouding.

Naturally the cardiac waist does not completely fill as in mitral valve lesions since the left ventricular arc and the aortic knob project markedly to the left.

described on page 151 appear in both oblique views A typical aortic configuration from simple dilatation due to increased resistance and left ventricular hypertrophy need not result.

Usually, typical aortic configuration occurs only when transverse expansion of the left ventricle is added to the lengthening and hypertrophy Signs of decompensation may be completely absent despite considerable cardiac enlargement as long as the dilatation is confined to the left ventricle. Morphologically the roentgen findings resemble those of aortic valve regurgitation

The left ventricular arc may also show slow, almost laborious systolic contractions as the roentgen correlate of pulsus tardus. Since, however, nonocclusion of the aortic valves frequently coexists and tends to produce large and strong pulsations, these characteristic slow pulsations are uncommon

Elongation, marked outward bulging, and a rounded angulation (fig. 112) of the right border of the vascular band are relatively common and indicate dilatation of the ascending aorta (Vaquez and Bordet) This poststenotic dilatation of the aorta is produced by the impact of blood ejected with greater force against the vessel wall from the hypertrophied left ventricle through the narrowed aortic orifice (Volhard). As a dynamic dilatation (p 371) it is not demonstrable at necropsy, if the valve lesion lasts for a long time the dilatation of the supravalvular aorta becomes anatomically fixed

Since failure of the left ventricle is followed by retrograde stasis in the left atrium and pulmonary circulation, the cardiac shadow and lung fields suffer the same changes as those described in the left heart failure of aortic valve regurgitation a mitralized aortic heart appears If, in the stage of mitralization, the characteristic pulsations of aortic stenosis or regurgitation are no longer evident (a very common event) a roentgenologic distinction of these two valvular lesions is no longer possible.

5 Tricuspid Regurgitation and Stenosis

Acquired pure tricuspid lesions are very rare Almost invariably lesions of this valve are combined with mitral or aortic valve disease Dressler and Fischer found involvement of the tricuspid in 30 per cent of valvular lesions due to rheumatic fever In one fourth of these the tricuspid orifice was stenotic. Relative incompetence of the valve resulting from right ventricular dilatation is much more common

Usually signs of a mitral or mitral-aortic lesion are present and only the right heart enlargement indicates with more or less probability that the tricuspid valves do not close or are narrowed

Tricuspid lesions produce no characteristic roentgen signs The difficulty in proof lies in the fact that alterations in size of the two sections of the right heart from incompetence or stenosis of the ostium vary extremely and usually differentiation between the atrium and ventricle is very imperfect (p. 156) Tricuspid regurgitation may be present with relatively little right heart enlargement and, on the other hand, it may be absent despite great enlargement. "Tricuspid configuration" (Holzmann), that is, elongation and a large laterally descending bulge of the right heart arc is certainly not pathognomonic of tricuspid regurgitation or stenosis (fig 113)

collection of pericardial fluid assists in enlarging the cardiac shadow; hydropericardium alone may cause very similar alterations of size and shape of the cardiac silhouette. A definite decision on the extent to which greater size of a mitralized aortic heart depends upon hydropericardium or actual cardiac enlargement is not possible. Consequently, reduction of the cardiac shadow after the circulation improves and after copious diuresis should always suggest, at first, the absorption of a pericardial effusion. It is, however, improper to conclude that the diminution always

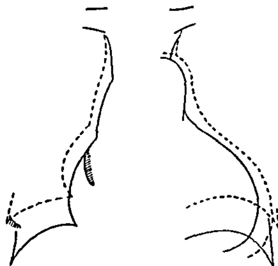


Fig. 110

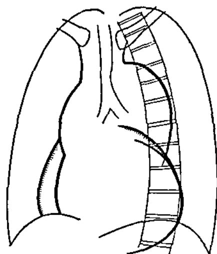


Fig 111

Fig 110 —Mitralized aortic heart in luetic aortic valve regurgitation, coronary stenosis, and *syphilitic aortitis Male, 55 years old Large cardiac shadow of aortic configuration,* broadened left and right, with markedly rounded left ventricular arc, shallow cardiac waist, the left atrium visible on the right, and signs of pulmonary passive congestion. Suggestion of pulsus celer on the aorta Six weeks later (orthodiagram in broken line) considerable dilatation of heart and small right *hydrothorax Necropsy syphilitic aortic valve regurgitation Severe syphilitic* aortitis with stenosis of coronary ostia Hypertrophy and dilatation of both ventricles, especially the left, with scars from myomalacia No pericardial effusion

Fig 111 —"Mitralized" aortic heart in left anterior oblique position (schematic) The enlargement of *the left* atrium is recognized on the upwardly ascending *left border of the cardiac shadow,* occasionally it is detected also when the border is subdivided into two arcs The enlargement of the right heart is expressed by the elongation and greater rounding of the right border of the cardiac shadow

or exclusively depends upon such absorption Unfortunately there is no way of reaching a reliable decision on this question.

4 Aortic Stenosis

The heightened resistance against which the left ventricle must work when the aortic ostium is stenotic may lead to its pure hypertrophy Roentgenologically this appears, at most, as greater rounding *of the left ventricular arc. Naturally signs of* dilatation due to increased resistance are very often added (p. 150), that is, the left ventricular arc elongates as well as becomes rounder. If this elongation involves both the inflow and outflow tracts of the ventricle, the changes in the cardiac shadow

like elevation (corresponding to analogous excursions described by Luisada and Fleischner on the left atrium in mitral regurgitation) is a tricuspid regurgitation very probable.

Pulsations along the great veins of the vascular band appear to offer better prospects in diagnosis If the right border of the vascular band can be sharply defined, occasionally on the screen a positive venous pulse may be observed in the superior vena cava and the right innominate vein (Groedel). Holzmann observed it in a patient whose engorged left innominate vein projected convexly above the aortic

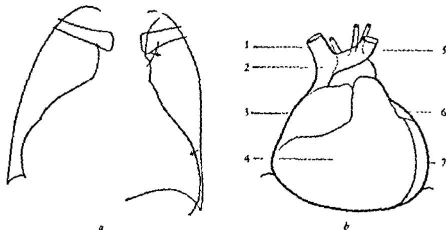


Fig. 114—Lesions of three valves with tricuspid regurgitation and stenosis and a positive venous pulse visible on the left innominate vein above the aortic knob Female, 52 years old (necropsy) The elongated, laterally descending right cardiac border, bulging far to the right, is subdivided into an atrial and ventricular arc No signs of pulmonary congestion (The arrows indicate the direction of the simultaneous pulsations on the left ventricular arc and the jutting shadow of the left innominate vein)

1. Right innominate vein
2. Superior vena cava
3. Right atrium
4. Right ventricle
5. Left innominate vein
6. Left atrium
7. Left ventricle

knob, we may add another instance (fig. 114) It is easier to obtain roentgenologic proof of a positive venous pulse in suitable cases by means of kymography.

Finally, reference should be made to the positive hepatic venous pulse sometimes recognized as a ventricular systolic, upward pulsation of the right diaphragm Hitzenberger recorded it for the first time by the roentgenkymograph. Holzmann observed a double hepatic pulse fluoroscopically in a patient with powerful pulsations of the right atrium

Unfortunately, these pulsatory phenomena, diagnostically important and clinically easy to recognize, are observed roentgenologically in only a minority of cases. The roentgenologic diagnosis of a tricuspid disturbance therefore must usually be based upon proof of right heart enlargement with relatively little or no pulmonary stasis, broadening of the superior vena cava, and upward displacement of the right diaphragm These signs gain in value, however, only when they appear successively in serial observations (fig 115).

Consequently other roentgen signs have been sought. Rösler correctly drew attention to the clarity of the lung fields, for both tricuspid stenosis and regurgitation unload the lesser circulation But, since the amount of pulmonary congestion depends upon still other factors, on the amount of circulating blood and on the power of the left heart for example, relative clarity of the lung fields cannot be regarded as proof of a tricuspid disturbance without further consideration. Naturally if the right heart is very large or enlarges progressively on serial examination, this assumption gains in probability.

Retrograde stasis, usually coexisting in the greater circulation, usually causes the vascular band to widen to the right by the increased pressure in the superior vena cava (Groedel) and upward displacement of the right diaphragm by the congested

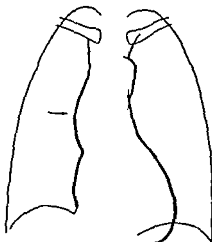


Fig 112

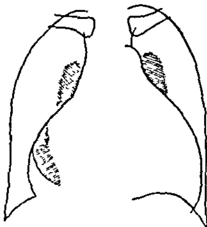


Fig 113

Fig 112.—Aortic stenosis (schematic) Elongation of the left ventricular arc Lengthening, *marked protrusion, and rounded angulation (arrow) of right vascular band owing to dilatation of the ascending aorta*

Fig 113.—Mitral-aortic valve lesion with particularly marked enlargement of the right heart but without signs of tricuspid regurgitation Woman, 58 years old There are signs of considerable pulmonary congestion

liver. Even these signs, by no means constant, can be evaluated only when they appear *with right heart enlargement. Frequently they are absent*

The signs which have been mentioned are common to tricuspid stenosis and regurgitation. Extreme stenosis is characterized by a particularly large right atrium and striking clarity of the lung fields. Occasionally excessive atrial enlargement also exists in the absence of a valve lesion (Dressler).

The pulsations of the right cardiac border, of the vascular band, and of the right diaphragm (liver) have attracted attention in the diagnosis of tricuspid regurgitation. Groedel considered lateral systolic ventricular pulsations of the right atrial arc pathognomonic Assmann, Dietlen, and Holzmann could not confirm this, this is comprehensible on the basis of roentgenkymograms of atrial pulsations (Zdansky and Ellinger) because the atrium normally enlarges during ventricular systole. Only if the right cardiac border shows, in the electrokymogram, systolic plateau-

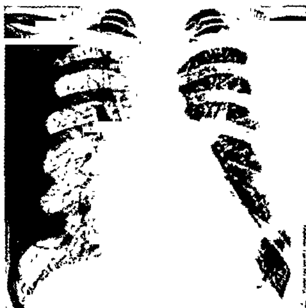


FIG 116.—Combined mitral-aortic valve lesion Considerable enlargement of cardiac shadow which is broadened to left and right with filling of cardiac waist and an elongated, markedly rounded left ventricular arc which showed large pulsations The large left atrium projects to the right over the right atrium The lung fields are clear although the hilar shadows are enlarged and lung markings are accentuated The aorta was normal in width and did not show a pulsus celer.

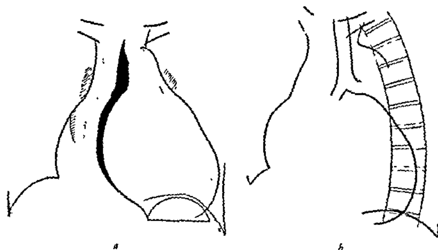


FIG 117 – Lesion of three valves (regurgitation and stenosis of aortic, mitral, and tricuspid ostia) from recurrent rheumatic fever. Female, 27 years old (necropsy) (a) Anterior view, (b) left anterior oblique view. Six years earlier (dotted line orthodiagram) no signs of a tricuspid lesion

It is impossible to differentiate with certainty tricuspid regurgitation from stenosis if pulsatory phenomena favoring the first are not unequivocal. It is also impossible to determine whether the regurgitation is relative or the result of rheumatic fever.

11. Rosler described an interesting relative tricuspid regurgitation. A 43 year old man developed cardiac failure with cyanosis, a positive venous and hepatic pulse and auricular fibrillation in the course of fourteen years. Roentgenologically, a very large cardiac shadow bulged to the left and right with a well preserved cardiac waist, marked widening of the superior vena caval shadow, and

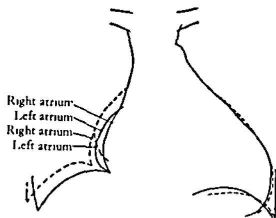


FIG. 115 —Increasing dilatation of right heart with development of relative tricuspid regurgitation. Female, 26 years old On March 27, 1935, the large left atrium extended about 7 mm. to the right (orthodiagram in solid line). On April 8, 1935, with a decrease in diuresis and gain in weight, there was considerable cardiac enlargement of the cardiac shadow to the right and a higher right leaf of the diaphragm (hepatic congestion) The left atrium at this time is seen within the right cardiac border (orthodiagram in broken line)

no pulmonary congestion Necropsy revealed relative tricuspid regurgitation resulting from massive dilatation of the right ventricle and atrium which the observer ascribed to myocarditis

6 *Combined Valvular Lesions*

The frequent combination of mitral and aortic valvulitis often causes hearts to display signs of mitral and aortic configuration (figs 116 and 117). According to the type and degree of combined valve lesions and according to conditions in the greater and lesser circulations, single sections of the heart experience very different changes; in some cases mitral, in others aortic configuration dominates the picture.

Mitral stenosis and aortic regurgitation to some extent exert opposite effects The small amount of blood admitted by the stenotic mitral ostium hinders left ventricular dilatation and hypertrophy that would occur with pure aortic valve regurgitation. When filling is diminished, the small stroke volume of the left ventricle usually dampens the large pulsations of the left ventricular arc and the appearance of aortic pulsus celer. As a rule, the aorta is normal in width or, owing to diminished filling, somewhat narrow. Consequently the heart displays the signs of a mitral lesion while the coexisting aortic valve regurgitation remains in the background or does not emerge (fig 118). The same also holds for the combination of mitral and aortic stenosis.

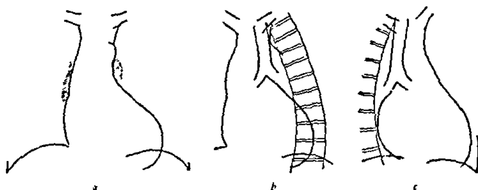


FIG 119.—Cardiac shadow of aortic configuration in regurgitation and stenosis of mitral and aortic ostia Male, 25 years old (*a*) Anterior view, (*b*) left anterior oblique view, (*c*) right anterior oblique view Only the enlargement of the markedly pulsating hilar shadows and the accentuated bulge of the posterior cardiac wall make it probable that a lesion of the mitral valve exists in addition to an aortic valve lesion

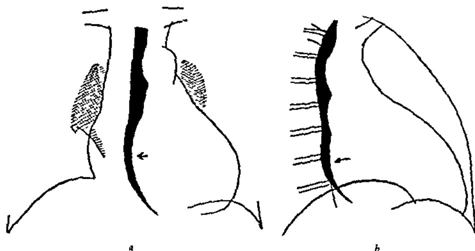


FIG. 120 —Typical picture of a mitral-aortic lesion Male, 56 years old. Necropsy aortic [illegible]

ventricular arc, rounded apex, and filling of the cardiac waist by the flatly curved, *protruding* pulmonary arc. The left atrium, considerably enlarged, forms the border on the right and displaces the esophagus to the right and posteriorly Signs of pulmonary congestion

In other cases the left ventricle fills enough to provoke hypertrophy and dilatation and to cause noteworthy enlargement of the stroke volume and pulsus celer. Then, despite elongation and greater rounding of the left ventricular arc, the cardiac shadow retains predominantly a mitral configuration and the picture resembles that of mitral regurgitation. Only the large pulsations of the left ventricular arc, aortic pulsus celer, so often present, and the distinctly prominent aortic knob hint at the combination with an aortic valve regurgitation. Moreover, similar alterations, except for the pulsatory phenomena, can also occur from mitral stenosis and coexistent hypertension

Furthermore, in the combination of a mitral-aortic lesion with pulmonary emphysema, the mitral configuration may dominate sufficiently to suggest a pure mitral lesion However, signs of marked left ventricular hypertrophy and dilatation as

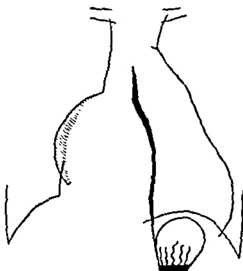


Fig 118 —*Cardiac shadow of mitral configuration in regurgitation and stenosis of mitral and aortic ostia* Female, 23 years old (necropsy) Very large left atrium projecting far to the right, displacing the esophagus to the left posteriorly.

well as distinct projection of the aortic knob should at least arouse the suspicion that no pure mitral lesion with emphysema exists but rather a combination with aortic valve regurgitation This conjecture becomes practically certain if pulsations of striking size are seen along the left ventricular arc and a pulsus celer. The absence of pulsus celer does not, however, preclude aortic valve regurgitation (p 186).

In contrast to the cases just mentioned, the aortic configuration in others dominates over the mitral (fig 119) or the picture of a mitralized aortic heart is simulated (fig. 120). The latter is comprehensible since cardiac dynamics are disturbed in the same direction in a mitral-aortic lesion and in a mitralized aortic heart. In one, the mitral valve is incompetent owing to shrinkage of the valve leaflets and in the other by stretching of the valve ring, in both, the left atrium enlarges, pressure rises in the pulmonary circulation, and the right ventricle dilates, in short, the morphologic results are analogous.

walls Assmann and Dietlen described cases of this kind. Dietlen stressed how difficult it is, clinically and roentgenologically, to distinguish this situation from a hydropericardium. Roentgenologic differentiation is rendered more difficult since the pulsations of these hearts are often very small and pulmonary congestion may be absent. On the basis of a case in which the diagnosis was established by paracentesis Dietlen suggested that the differentiation from hydropericardium is possible when the enlarged left atrium is seen within the right cardiac border as a double shadow and the inferior vena cava fills the lowered right cardiodiaphragmatic angle.

7 Pulmonary Regurgitation after Endocarditis

In contrast to relative pulmonary regurgitation (p 178f) pure pulmonary regurgitation from endocarditis is rare (Weinberger) Usually it leads to exclusive or predominant dilatation and hypertrophy of the right ventricle by diastolic overloading This produces a mitral configuration with the heart broadened mainly to the left, on its protuberant pulmonary arc, a pulsation like that of pulsus celer is seen and the enlarged hilar shadows show intrinsic pulsations Both cardiac borders display large excursions which arise from the right ventricle Naturally, signs of pulmonary congestion are absent. Usually the left atrium is normal

In the anterior view, a morphologic differentiation from a relative pulmonary insufficiency (Graham Steell) is impossible although the large pulsations like those of pulsus celer on the pulmonary arc and the hilar shadows make the latter seem improbable When the cardiac shadow enlarges as the right heart begins to fail and as a consequence of the smaller stroke volume, the large pulsations of the heart and of the hilar shadows vanish and the picture more nearly resembles that of relative pulmonary regurgitation Only the normal size of the left atrium allows one to exclude a Graham Steell relative pulmonary regurgitation But if this is established there remains the possibility of a congenital defect of the atrial septum (p 264), in which blood passes out of the left heart into the right It is impossible to reach a definite decision by x-ray. Since pulmonary regurgitation of inflammatory origin is relatively rare, one should think first of the congenital anomaly just mentioned.

II. Hypertension

A heart with normal functional capacity can, as was explained on page 117, overcome increased peripheral resistance by more powerful contractions which gradually lead to hypertrophy of its walls In time, however, systolic emptying of the left ventricle becomes imperfect and causes dilatation due to increased resistance (p 138), this enables the ventricle to perform the added work and is also followed by mural thickening The roentgen picture of various forms of hypertension is uniform There are signs of hypertrophy and, should the opportunity arise, of left ventricular dilatation in all grades from the minimal and scarcely recognizable to the most extreme

Although no specific picture should be expected in the different forms of hypertension, differences occur in the extent of hypertrophy and dilatation, although these

Despite these similarities, some differences permit the distinction between a mitral-aortic lesion and a mitralized aortic heart with more or less probability. These points are summarized below.

Mitral-aortic lesion	Mitralized aortic heart
Left atrium markedly enlarged projecting decidedly into the posterior mediastinum and often forming the right cardiac border.	Left atrium less enlarged, therefore projecting less into the posterior mediastinum, on the right, it is visible within the cardiac shadow and occasionally as a double shadow but usually not projecting beyond the right atrium.
Pulmonary arc and often conus pulmonalis definable as a prominent arc projecting into the cardiac waist	Cardiac waist merely shallow Pulmonary arc and conus pulmonalis not bulging
In compensation, merely enlarged hilar shadows, often with intrinsic pulsations Signs of pulmonary stasis only in decompensation	Almost always more or less severe pulmonary congestion.
In compensation sometimes only a moderately enlarged heart, occasionally almost normal in size	Usually the heart is considerably enlarged

These differential signs depend largely on the fact that in combined lesions, retrograde stasis in the left atrium and consequent reduction of pressure potential in the pulmonary circuit develops very gradually, leading to marked, sometimes enormous, left atrial enlargement (fig 118) as well as to right ventricular hypertrophy and dilatation with their results, so-called mitralization of the aortic heart on the other hand is a relatively acute event which happens with left ventricular failure. Consequently, with the mitralization, left atrial dilatation usually remains within modest limits (Dietlen) so that real right ventricular hypertrophy and marked dilatation of the pulmonary artery usually do not emerge. On the other hand pulmonary congestion is more or less marked, as a rule, as long as the right heart also has not failed. The differential signs have exceptions and, at times, no distinction can be made between relative mitral regurgitation and the form following rheumatic fever (Dietlen, Vaquez and Bordet, Henschen).

The heart with a combined mitral-aortic lesion may closely resemble a hypertensive heart with complicating emphysema, in both, the cardiac waist may be filled by the protruding pulmonary artery and conus pulmonalis, in both, hilar shadows are enlarged and often show intrinsic pulsations. Differentiation is possible, however, since in combined lesions the left atrium is enlarged while in a compensated hypertension-emphysema it is normal in size If the left ventricle fails in a patient with hypertension and emphysema, differentiation from a mitral-aortic lesion may be impossible since now left atrial enlargement is present

With lesions of three valves the heart is usually considerably enlarged and, as a [illegible] (fig 114). No definite decision on the [illegible] ole (p 194).

[illegible] sion the large globular heart bulges far to the left and right below a short vascular band. The markedly rounded borders may merge into single arcs and may extend almost to the lateral thoracic

shadow enlarges exclusively or predominantly to the left or universally. The left cardiac border elongates and bulges more roundly to the left, indicating left ventricular dilatation and hypertrophy, often the cardiac waist is somewhat shallow. The aorta dilates little or not at all The pulsations of the cardiac shadow are often distinctly diminished

Commonly, in acute nephritis, the diaphragm is elevated (Zdansky) but this is not always striking, often this is recognized only when the diaphragm descends after the renal process subsides Very probably it results from retention of fluid by the abdominal viscera (fig 122). Naturally this elevation of the diaphragm may displace the heart upward and transversely, a point to be considered in decisions on cardiac size

Occasionally the only roentgenologic sign of acute nephritis, but one rarely missed, is bilateral hydrothorax (figs 121 and 122), in many cases the amounts involved are small so that they may escape clinical detection Sometimes they just fill the costophrenic angles or appear as a small seam, shifting on respiration, and as a lamellar shadow, extending up along the thoracic wall from the costophrenic angles. These small collections of pleural fluid have diagnostic significance because they appear regularly (Eppinger). They have no connection with cardiac stasis but are nephritic exudates

Frequently, in acute nephritis, the lung fields show striking changes the hilar shadows are enlarged, compact, hazy, and surrounded by linear and focal shadows These densities may be confined to the perihilar areas but often include large sections of the middle and basal parts of both lungs, occasionally they completely fill both lung fields, obscuring them by more or less intense but not homogenous haze If these densities are confined to the central parts of the lungs, the absence of accentuated vessel shadows in the peripheral parts of the fields indicates that pulmonary congestion of cardiac origin is not present. Actually pulmonary edema of nephritic origin exists, at one time this (p. 350) is mainly interstitial (fig. 253) and at another parenchymatous (figs 251, 252). Often it develops very insidiously and escapes clinical detection for a long time, particularly when it is largely interstitial and merely involves the central parts of the lungs Therefore, its roentgenologic detection has great practical importance (Zdansky). It can vanish completely in a few days

Naturally, pulmonary stasis of cardiac origin also occurs at times Occasionally one finds an enlarged heart of aortic configuration showing signs of mitralization

The enlarged cardiac shadow in acute nephritis may become smaller. Often, in a amazingly short time, within a few days, even within twenty-four hours, the enlargement disappears. This raises the question as to whether the heart actually enlarges, or a pericardial effusion forms, or both. Since it is impossible on the basis of a roentgenogram to exclude a pericardial effusion, even one containing several hundred cubic centimeters (p 326), great caution must be observed in statements about actual increase of cardiac size. This holds particularly since bilateral pleural effusion is almost invariably present and this makes a corresponding collection of pericardial fluid very probable When it is considered that accumulations of pleural fluid are usually small while the cardiac enlargement may be considerable, one must assume that in such cases cardiac dilatation is present or that much more fluid is

lack decisive value, they have diagnostic and prognostic importance within the frame of the complete clinical picture and often significance for therapy. Clinicians and pathologists, like v. Romberg, Volhard, Fahr, Passler, Jores, Kroetz, and others, have drawn attention to these differences.

1. Nephritic Hypertension

Often in acute nephritis the cardiac shadow is entirely normal in size and shape when the systolic pressure reaches 160–180 mm. Hg or more. Hypertrophy of the normally functioning left ventricle and the dilatation due to increased resistance

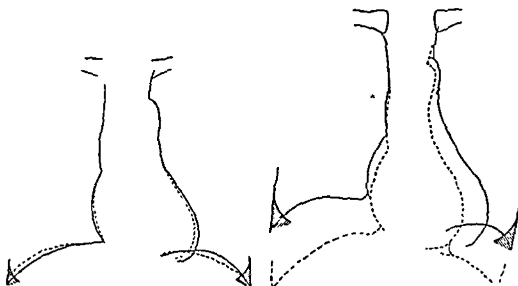


Fig. 121 Fig 122

Fig 121.—Slight cardiac enlargement and bilateral small hydrothorax in acute nephritis Male, 25 years old ——— November 27, 1936 blood pressure 200/150 mm Hg, - - - - - January 4, 1937 blood pressure 130/95 mm Hg The bilateral hydrothorax has disappeared

Fig 122 —*Moderate enlargement of cardiac shadow, bilateral small hydrothorax, and elevation of diaphragm in acute nephritis Recession of all signs after the nephritis vanished* Male, 32 years old ——— February 6, 1935 *blood pressure* 150/60 mm *Hg*, NPN 85 4 mg per cent, - - - - - *April 20, 1935 blood pressure 115/48 mm Hg, NPN 50 4 mg per cent*

(p 138), often present, may be so trifling that no unequivocal alteration of the cardiac shadow occurs (p. 149). Naturally if there is an occasion to reexamine the heart of a patient with nephritis after the process has subsided and the blood pressure has returned to normal, a comparison of the orthodiagrams or teleroentgenograms usually shows that during the illness the arc of the left ventricle was somewhat elongated and rounded (figs. 121 and 122). These trifling alterations reflect a longitudinal

In many cases of acute nephritis the cardiac shadow is enlarged and this may considerable (Moritz, Dietlen) A connection with the blood pressure level, as Groedel originally observed, is common but not always apparent. The cardiac

heavy labor, obesity) are added to the chronic renal malady. Then, a large aortic heart, widened particularly to the left, develops with its massive left ventricular arc sometimes extending almost to the left chest wall and with small excursions of its borders. Analysis of the roentgenogram reveals left ventricular hypertrophy and dilatation and usually a dilated aorta. Often signs of mitralization are noted. shallow cardiac waist, enlarged hilar shadows, increased lung markings, and an enlarged left atrium, that is, alterations indicating left ventricular failure. Finally, the right heart also attains considerable size as expressed by increased widening to the right, greater bulging of the anterior cardiac wall in the left anterior oblique position, and, often, light lung fields.

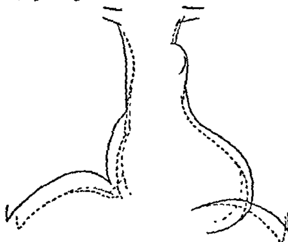


FIG 123 —Progressive reduction of cardiac size and restoration of cardiac compensation after strophanthin despite persistent elevation of NPN in a case of contracted kidney Death from cerebral hemorrhage eight days after final observation Male, 55 years old (necropsy). ———February 1, 1937 blood pressure 205/125 mm Hg, NPN 86 8 mg per cent, pulmonary congestion, - - - - - - February 11, 1937 blood pressure 210/115 mm Hg, NPN 108 mg per cent, no pulmonary congestion - . - . - . - February 15, 1937 blood pressure 210/115 mm Hg, NPN 106 mg per cent, no pulmonary congestion

This pure cardiac decompensation is not the typical end stage of nephritis and is actually rather rare (v Romberg)

cardial damage This is not intended to imply that the retention of nitrogenous products leads to cardiac dilatation nor that dilatation parallels such retention Frequently, the heart becomes smaller when its strength is improved by digitalis despite a high level of residual nitrogen (fig 123)

There is also no constant relation between the height of the blood pressure and cardiac size, a certain parallelism is seen in single cases for the heart tends to become larger or smaller as pressure rises or falls (fig. 124).

At all events, examination very frequently discloses considerable cardiac dilatation in the end stage of secondary contracted kidney. The opposite opinion has been expressed repeatedly for the following reason. usually the stage of cardiac dilatation

excreted into the pericardial than into the pleural cavity. Although this possibility cannot be summarily dismissed, it loses plausibility since there are adequate reasons for the appearance of cardiac dilatation when renal failure occurs. Increased blood volume, elevation of peripheral resistance and uremic-toxic damage of the myocardium undoubtedly tend to produce progressive cardiac dilatation. Actually, even in this stage of the disease, clinical signs of cardiac failure are common and necropsy may reveal cardiac dilatation predominantly involving the left ventricle.

Undoubtedly one must consider the possibility of cardiac dilatation in the acute stage of nephritis; Volhard, without hesitation, interprets the enlargement of the cardiac shadow in these cases as a result of dilatation. On the other hand, Alwens and Moog believe that the enlargement depends only to a slight extent on cardiac dilatation while a hydropericardium is mainly responsible. Their opinion is based on the fact that the cardiac shadow becomes smaller coincident with the disappearance of the pleural effusions, pulmonary congestion, and edema. This argument is not entirely convincing because it is just as reasonable to assume that the heart can also become smaller with falling blood pressure, removal of fluid, and recession of uremia. We have frequently made an observation of interest the cardiac shadow increases in size with undiminished pulsations and persistence of the individual arcs when signs of renal failure appear, particularly as the nonprotein nitrogen rises, since this enlargement may vanish when residual nitrogen level falls, this seems to suggest that cardiac enlargement, at least in many cases, indicates dilatation from an uremic-toxic myocardial injury. Naturally this does not preclude a pericardial effusion from contributing to further enlargement of the cardiac shadow. To what extent this happens cannot be stated owing to the impossibility of recognizing small pericardial effusions roentgenologically. Consequently, while it cannot be denied, despite the probability, that increased size of the shadow often depends upon an actual cardiac enlargement, nothing definite is known about the frequency and extent of it. Conclusions on this score will be obtained only when pericardial paracentesis is performed systematically.

In the second or continued stage of nephritis (Volhard) the cardiac shadow is enlarged slightly or not at all. Among patients with war nephritis, Franke-Musils found a normal cardiac shadow in 10.4 per cent and pure left ventricular hypertrophy in 45 per cent, the heart was dilated in 29 per cent, the left ventricle in 11 per cent, and both ventricles in 18 per cent. In our experience, the hypertrophy and, usually minor, dilatation is limited to the left ventricle, the left atrium and the right heart usually remain normal Extreme cardiac enlargement like that of acute nephritis and terminal renal failure is relatively uncommon in the continued stage of nephritis The lung fields are clear and without signs of pulmonary congestion or edema

Volhard stressed that the degree of left ventricular hypertrophy and dilatation, in general, depends upon the height and, to a certain measure, upon the duration of hypertension; the constitution and requirements of the body musculature could also influence the extent of left ventricular changes Actually, older individuals who have suffered for years, and particularly slender women, often have hearts of entirely normal size Tallquist and Hess emphasized the significance of constitution in the absence of cardiac hypertrophy. On the other side, occasionally decided dilatation appears with phenomena of renal failure, if other injuries (excessive use of beer,

marked left ventricular hypertrophy and dilatation is definitely greater (Volhard, Fahr). According to Lange and Wehner, in fixed hypertension with cardiac compensation, the average widening of the heart to the left parallels the height of the blood pressure This holds, however, only for average values in large series, in single cases there are many exceptions to this rule Very commonly, patients with a systolic blood pressure of 200 mm Hg and more do not show alterations of the heart which are demonstrable roentgenologically, but this also happens in patients with labile hypertension *when the high values are only momentary or shortlasting* (fig. 125), this agrees with clinical and pathologic experience. Similar observations have been made in the hypertension of chronic lead poisoning (Vaquez and Bordet). As in nephritic hypertension, the morphologic alterations to which the heart is exposed depend not only on the height and duration of hypertension but also on constitutional factors (Tallquist, Hess) and conditional influences such as the mode of life, alcoholism, and obesity A mode of living involving physical and mental strain and abuse of alcohol, tends to dilate an already overburdened heart, a quiet life, mental tranquility, old age, rest enforced by other diseases, emaciation, and many other influences offset cardiac hypertrophy and dilatation (Volhard) It is peculiar to the nature of the disease that factors which promote cardiac enlargement frequently are relatively more effective in hypertensives than in patients with nephritis This is clear even from the high incidence of large hearts in hypertensive individuals *More specifically, hypertensive patients have a remarkably high incidence of* coronary sclerosis contributing to left ventricular dilatation Averbuck found severe coronary sclerosis in 25 per cent of hypertensive patients dying of cardiac decompensation

While extreme cardiac dilatation in nephritis causes one to think primarily of uremic-toxic damage, the dilatation of essential hypertension usually involves a myocardial lesion conditioned by coronary sclerosis and often latent for years Naturally patients with nephritis may also develop coronary sclerosis and in hypertension the heart may also be injured by uremic-toxic influences. These facts must be born in mind in evaluating a roentgenogram Acute dilatation of hypertensive hearts can recede more or less completely just as in nephritis (fig 126) Here also, one must remember that the absorption of a pericardial effusion may simulate a reduction of cardiac size

The size of the heart in hypertension has only limited prognostic value (Vaquez and Bordet) Often large hearts remain unchanged and perform capably for years without manifestations of decompensation, on the other hand, relatively small hearts may fail prematurely

In a majority of cases the aorta is diffusely widened Sometimes dilatation occurs mainly in the initial portion in the absence of lues (Schlesinger) Visible atheromatous deposits of calcium are common, especially in the arch The report (Lange and Wehner) that the aorta may be elongated but slender in hypertensives is not reliable although in young patients and in slender, even older, individuals, occasionally the aortic diameters may be normal.

In general, the picture found in hypertension can be distinguished from that of aortic valve regurgitation only by the absence of large pulsations on the left cardiac border and pulsus celer along the aortic shadow (Dietlen)

is rather short and at necropsy the size of the heart during life cannot be accurately estimated since ordinarily it shows maximal contraction after death

Both halves of the heart enlarge although that of the left ventricle dominates by far. Enlargement of the right heart without failure of the left has been properly denied by Passler and Kirch

Precisely in the end stage of nephritis, one must always count on the possibility of a pericardial effusion simulating cardiac enlargement or at least contributing to an enlarged cardiac shadow.

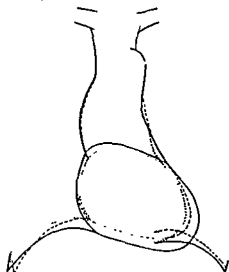




FIG 124 FIG 125

FIG 124.—Alterations of cardiac size in pyelonephritic contracted kidney which approximately *followed fluctuations of the blood pressure* ______ *October 14, 1936, blood pressure 175/95* mm Hg, NPN 106 4 mg per cent, ... December 4, 1936 blood pressure 140/85 mm Hg, NPN 124 9 mg per cent, -.-.- January 18, 1937 blood pressure 185/85 mm Hg NPN 95 2 mg per cent The heart is not essentially enlarged despite the elevated NPN Its size changed with fluctuations of the blood pressure The ascending aorta is dynamically dilated as may be seen by the angular *rounding of ascending aorta (necropsy)*

FIG. 125 —Cardiac shadow of normal size without signs of left sided hypertrophy in essential hypertension with repeated cerebral vascular accidents Blood pressure, 205/120 mm Hg Slightly built, asthenic female, 52 years old

As in subacute renal failure, a central pulmonary edema often develops insidiously or a more acute widespread type appears In some cases pulmonary congestion results from failure of the left heart.

In a roentgenogram of the heart taken after death from uremia, Geipel demonstrated calcification of the myocardial fibers in the form of delicate, more or less compact, curved and wavy shadows which corresponded in arrangement to the course of the muscle fibers Apparently this has not been found in vivo.

2 *Essential Hypertension*

Since essential hypertension places demands upon the heart analogous to nephritis, in both instances alterations of the cardiac shadow are fundamentally the same. But, in contrast to nephritis, it is generally recognized that the incidence of

limp (Lange and Wehner). From this cardiac shadow a slender long vascular band rises to a high aortic knob which tends to project decidedly to the left. Vaquez and Bordet made the excellent comparison of this picture to a phrygian miter (figs. 128 and 129).



FIG. 128 FIG 129

FIG 128 —"Senile heart" Male, 60 years old Moderately enlarged heart, transverse owing to atheromatous aortic elongation

FIG 129.—"Senile heart" Female, 80 years old, with a blood pressure of 205/90 mm. Hg The heart is normal in size but has become transverse owing to elongation of the somewhat dilated aorta There is increased rounding of the left ventricular arc

III. The Myocardial Injuries

The various myocardial injuries create no pathognomonic roentgen picture The gross morphologic alterations produced by myocardial damage depend not only on the type of injury but also to a great extent on the state of the heart before the injury and extracardiac circulatory conditions after its appearance Consequently, similar injuries may produce very different effects while diversified damages may lead to similar changes in cardiac size and shape. Above all, not every myocardial lesion causes cardiac dilatation As cardiac strength declines, the circulation may be transformed in such a way that the heart is relieved and discharges the reduced demands without increase of size and change of shape This explains the common observation that even severely damaged hearts often are not enlarged, indeed they may even be small, and that hearts about to fail owing to a severe coronary artery disease may show an entirely normal roentgenogram. Moreover, small hearts are found in myocarditis, in some cases of severe thyrotoxicosis and in Addison's disease. Therefore, normal size and shape of the cardiac shadow does not exclude a myocardial lesion.

On the other hand, dilatation of the heart, roentgenologically demonstrable, usually indicates myocardial damage except when extreme demands on cardiac strength or abnormal working conditions have induced dilatation, as in the left ventricle in mitral regurgitation for example.

1 Myocarditis and Infectious-Toxic Lesions of the Heart

Myocarditis is extremely common. In addition to the rheumatic myocarditis accompanying every acute polyarthritis, inflammatory lesions are by no means unusual in respiratory infections, after sore throat and the like Myocarditis is common in diphtheria, scarlet fever, typhoid fever and typhus, pneumonia, and malaria, as well as many other severe infections

Often the clinical phenomena of the myocardial injuries are neither striking nor characteristic Commonly, the myocarditis is entirely unnoticed Fever, feeling of ill-health, palpitation, precordial pain, anorexia, and pallor are more or less frequent but by no means regular manifestations. Usually no enlargement of the heart is detected clinically in uncomplicated myocardial inflammation, moreover, auscultation yields no characteristic findings Usually the sounds are pure except for a

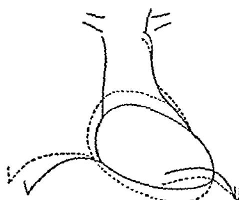


FIG 130 —"Latent dilatation" of the heart in endomyocarditis Female, 37 years old Electrocardiogram prolonged conduction time (0 27 second) On standing (orthodiagram with solid line) the cardiac shadow is normal in size but rests somewhat broadly and low on the diaphragm In the recumbent position (orthodiagram with broken line) the heart is dilated

systolic murmur at the apex Often the rate is normal, more rarely it is increased On the other hand slight exertion often causes a disproportionate increase of rate Alterations are found in the electrocardiogram only when foci of inflammation are large or are located in the conduction system.

The roentgenogram also is not characteristic The cardiac shadow may be normal irrespective of the etiology of the myocardial injury

Often it is striking that the cardiac shadow rests low and broad on the diaphragm creating the impression that the heart is flaccid (fig 130). Frequently this impression is heightened by lability of the cardiac shape with respiration and positional alterations of diaphragmatic level. This lability of shape is often regarded a sign of reduced myocardial tonus (p 110) and this assumption has some basis in fact Nevertheless, it should not be forgotten that many of these patients have a peripheral vasomotor weakness as indicated by their remarkable susceptibility to collapse At least in the erect position this vasomotor weakness diminishes cardiac filling to which the heart must adapt itself by change of shape (and size) (pp. 93, 102) In hypertrophied hearts this lability of shape is usually missed (Dietlen)

More important than this lability of cardiac shape is the alteration of its size. It should be mentioned once again that even severe myocardial damage does not necessarily cause cardiac enlargement. If, however, one uses every opportunity to

limp (Lange and Wehner). From this cardiac shadow a slender long vascular band rises to a high aortic knob which tends to project decidedly to the left. Vaquez and Bordet made the excellent comparison of this picture to a phrygian miter (figs 128 and 129).



FIG 128 FIG 129

FIG 128 —"Senile heart" Male, 80 years old Moderately enlarged heart, transverse owing to atheromatous aortic elongation

FIG 129 —"Senile heart" Female, 80 years old, with a blood pressure of 205/90 mm Hg The heart is normal in size but has become transverse owing to elongation of the somewhat dilated aorta There is increased rounding of the left ventricular arc

III. The Myocardial Injuries

The various myocardial injuries create no pathognomonic roentgen picture The gross morphologic alterations produced by myocardial damage depend not only on the type of injury but also to a great extent on the state of the heart before the injury and extracardiac circulatory conditions after its appearance Consequently, similar injuries may produce very different effects while diversified damages may lead to similar changes in cardiac size and shape Above all, not every myocardial lesion causes cardiac dilatation. As cardiac strength declines, the circulation may be transformed in such a way that the heart is relieved and discharges the reduced demands without increase of size and change of shape This explains the common observation that even severely damaged hearts often are not enlarged, indeed they may even be small, and that hearts about to fail owing to a severe coronary artery disease may show an entirely normal roentgenogram Moreover, small hearts are found in myocarditis, in some cases of severe thyrotoxicosis and in Addison's disease Therefore, normal size and shape of the cardiac shadow does not exclude a myocardial lesion

On the other hand, dilatation of the heart, roentgenologically demonstrable, usually indicates myocardial damage except when extreme demands on cardiac strength or abnormal working conditions have induced dilatation, as in the left ventricle in mitral regurgitation for example

the recognition of myocardial damage. Sometimes the latent dilatation represents a residual phase of a manifest dilatation which previously appeared even in the erect posture (fig. 133).

The reason for the latent appearance of dilatation in the erect position is found in the vasomotor weakness mentioned earlier; this allows a disproportionately large amount of blood to accumulate in the dependent parts of the body and diminishes cardiac filling (p. 93). To demonstrate true cardiac size, it is necessary to create optimal conditions for its filling and these are provided by the horizontal position. The significance of peripheral vasomotor weakness for the size of these hearts is evident from the fact that they may be enlarged in the recumbent patient during the disease but rather small on standing and indeed smaller than before the illness or after convalescence (fig 132). In short, comparative determinations of cardiac size in the standing and recumbent positions should never be neglected.

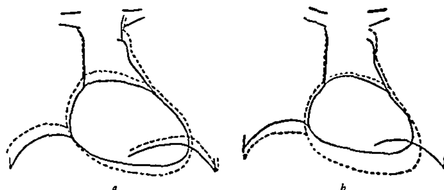


FIG 133.—Transition of manifest dilatation into latent dilatation in rheumatic myocarditis Male, 24 years old, with acute polyarthritis ——— Vertical orthodiagram, - - - - - horizontal orthodiagram (*a*) At the height of the disease the heart is considerably enlarged and this is even more apparent *in the recumbent position*, EKG, *normal*. (*b*) *Five weeks later, when subjectively* well, only latent dilatation is detected, EKG now shows prolonged conduction time (0 22 second)

Cardiac enlargement in chronic and recurrent diseases usually remains within modest limits. As a rule both halves of the heart are equally involved, as analysis of the cardiac shadow will reveal. Only with very severe and usually fatal myocarditis does the dilatation increase to a considerable degree. Signs of pulmonary congestion are usually missed but at times fluoroscopy demonstrates a beginning central pulmonary edema which perhaps escaped detection clinically.

The situation is different when cardiodynamics were altered by a valvular lesion or *hypertension* before the appearance of the myocardial injury. Under these circumstances, all sections of the heart do not as a rule, enlarge uniformly but dilatation occurs predominantly in those sections which were subject to the greatest demands; dominant dilatation of the left ventricle occurs *with aortic valve regurgitation or* hypertension of the left atrium and right ventricle in mitral stenosis and so forth (fig. 134). In this way, acute decompensation may occur with manifestations of stasis in the pulmonary or systemic circulation. With each new bout of myocarditis and each exacerbation of rheumatic fever such hearts may enlarge but they retain a given size and shape in the interim (fig 135).

examine all suspicious cases in the horizontal position, it is surprising how frequently such cardiac dilatation is found when it has been missed in the upright posture. This dilatation, manifest first in the recumbent posture and originally empasized by Dietlen, is called "latent dilatation." It indicates a myocardial lesion

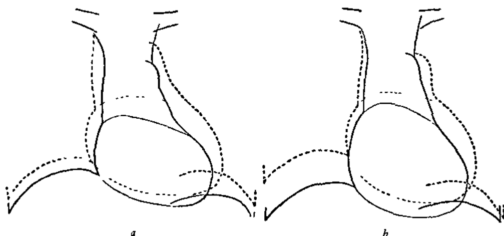


FIG 131 —Transition of "latent dilatation" into manifest dilatation also in erect position Male, 30 years old, *with acute polyarthritis and myocarditis* ——— *Vertical orthodiagram,* - - - *horizontal orthodiagram* (*a*) November 25, 1936, fever and joint swelling, EKG normal, the heart dilated only with examination in recumbent position (*b*) March 5, 1937, free from complaints, EKG conduction time prolonged Dilatation of the heart is now also manifest in erect position

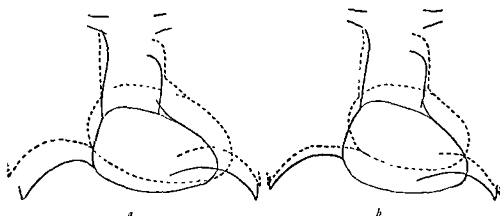


FIG 132 —Transient "latent dilatation" in rheumatic myocarditis Female with polyarthritis ——— Vertical orthodiagram, - - - - - horizontal orthodiagram At the height of the rheumatic fever (*a*) examination in recumbent position revealed cardiac dilatation Eighteen months later, with complete well-being (*b*) the latent dilatation is no longer present On the contrary, on standing the heart is somewhat larger than at the time of the illness This enlargement of vertical heart size may be ascribed to the disappearance of peripheral vasomotor weakness that caused orthostatic reduction of cardiac size at the height of the illness

We noted it not rarely when the electrocardiogram was normal or became abnormal only subsequently (fig. 131). Often we observed it transiently in acute polyarthritis (fig. 132), in sepsis, and even in acute tonsillitis, just as often we have seen its gradual evolution into a fixed dilatation (fig. 131). Its discovery has special value in

Whether the schematic arrangement of certain kymographic curves to different phases of chronic myocardial damage is valid, as Stumpf, Heckmann, and others tried to show, is open to question. According to them, the first stage is characterized *by a moderate increase of cardiac breadth and length with enlargement of the average* stroke amplitude over the value of about 5 mm., the second stage is revealed by general cardiac enlargement with decreased amplitude of movements at the apex while the average stroke amplitude returns to approximately normal values, the third stage is indicated by the universal decrease of movements, reduction of the movement area, and recession of the average stroke amplitude to values near 1.5 mm. (teleroentgenograms). It must be stressed however, that the visible pulsations of the cardiac shadow are determined by anatomic and functional factors, some of which lie within the muscle and others in the periphery, the combinations are too diversified to establish rigid rules for the pulsations The large lively pulsations, so frequent in myocardial injuries, for example, may depend upon a relaxation of anatomic origin, diminished diastolic tension of the myocardium, reduced inflow of blood into the heart, an accelerated flow of blood, or a combination of these factors Consequently, the pulsations are not characteristic of a definite type or even a definite grade of myocardial damage, rather they appear when the influx of blood is impaired in vasolabile individuals and enteroptotic subjects (Laurell), with fever and

of the left ventricular arc may show splintering, premature systolic median movements as well as lateral or even median plateau formations (Stumpf, Heckmann)

In our experience the flapping pulsations, conditioned by orthostatic reduction of inflow to the heart, frequently disappear in the horizontal position Perhaps this may have some diagnostic value.

In very rare cases necrotic muscle fibers or groups of muscle fibers may calcify, these may be roentgenologically demonstrable. In a man, 37 years old, who had suffered from rheumatic fever since the age of 8, Clark saw calcareous streaks within the cardiac shadow and ascribed them to myocardial calcification Baumann and Naumann demonstrated roentgenologically a calcified focal necrosis in the bundle of His (ventricular septum) in two cases of complete heart block (Adams-Stokes), *subsequently, necropsy confirmation was obtained.*

In summary, there are no roentgen findings specific for the diverse inflammatory or infectious-toxic injuries of the myocardium. In general acute cardiac enlargement, or enlargement of single sections appearing in the course of infectious-toxic diseases, suggests myocardial injury of the types mentioned Moreover, lability of cardiac form has a certain significance Special weight, however, is placed upon "latent dilatation" and for this reason examination in the recumbent position should never be omitted. Sometimes latent cardiac dilatation antedates electrocardiographic alterations. Lively pulsations of the cardiovascular shadow also have definite significance.

2 The Heart in Anemia

This term refers merely to the heart in severe chronic anemias, to pernicious as well as the secondary anemias. Ludke and Schuller, as well as Meyer and Seyder-

Cardiac dilatation of inflammatory as well as of toxic origin may recede greatly Often the acutely enlarged heart regains its former size and shape within a short time. On the other hand, a pericardial effusion may simulate such transient enlargement (Dietlen). Some have even gone so far as to suggest that a reduction of volume, roentgenologically observed, in the presence of a valvular lesion almost proves the absorption of an unrecognized pericardial effusion. This is not valid as a broad generalization Certainly it is difficult, if not impossible, to exclude a pericardial effusion absolutely; but, in many cases careful analysis of the roentgenogram brings

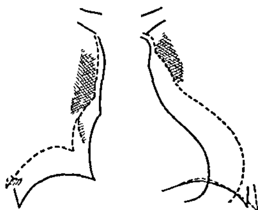


Fig 134

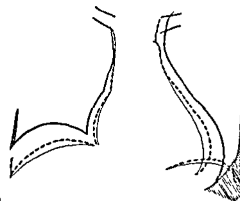


Fig. 135

Fig 134.—Cardiac shadow of normal size and shape in a clinically unequivocal mitral-aortic lesion. Recurrent endocarditis Female, 22 years old (necropsy) Even in the oblique position no left atrial enlargement demonstrable Only the very lively pulsations of the cardiovascular shadow indicated myocardial damage. Seven months later (orthodiagram in broken line), after another recurrence, there was cardiac dilatation and the typical picture of a combined lesion

Fig 135.—Endomyocarditis in acute rheumatic fever Development of a mitral lesion Female 22 years old The woman, previously healthy, became ill 14 days earlier from fever and painful swollen joints ———March 9, 1936 roentgen findings normal, no clinical signs of valvular lesion, EKG prolonged conduction time (0 22 second), ———April 9, 1936, cardiac shadow considerably enlarged, mitral configuration, lively pulsations, left atrium enlarged, bilateral small hydrothorax, clinical signs of mitral lesion, EKG no T wave in leads I and II, -- --May 14, 1936 patient afebrile and free from complaints, cardiac shadow essentially smaller but still shows mitral configuration, left atrium enlarged, EKG normal

evidence that the cardiac enlargement is produced by an unequal increase of single sections, perhaps of the right or left ventricle or left atrium

to Stumpf, the excursions decrease particularly in the apical section of the left cardiac border so that here they are smaller than in the basal area (p. 55). Often, on the contrary, pulsations on the left cardiac border are, on the whole, enlarged (Bordet, Holst, Stumpf); then, not rarely, they are very lively, almost flapping in character ("excited type," Dietlen). Especially when aortic valve regurgitation coexists, the height of excursions along the left ventricular arc and of the vascular band, already enlarged, increase remarkably, excursions of 4 to 5 mm. in height and more are not uncommon.

anemia that the systolic and diastolic limbs of the ventricular kymogram have a concave and not a convex course as would be characteristic of myocardial damage in his opinion.

The roentgen picture does not reveal to what extent cardiac enlargement depends upon anemic myocardial damage, upon greater circulating blood volume, or upon hypertrophy of the muscle (Grunberg, Porter).

As mentioned above, the cardiac enlargement does not parallel the degree or duration of the anemia. Often with primary or secondary anemia the heart remains *completely normal in size. Enlargement is missed almost regularly when the anemia* is a single manifestation of a cachexia with accompanying dehydration (cachexia of malignancy, intestinal intoxications, pulmonary or intestinal tuberculosis). In these cases the diminished blood volume obviously offsets the cardiac enlargement.

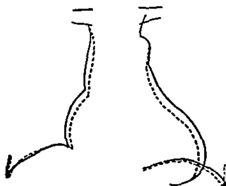


FIG 137.—Rapid reduction of heart size after recession of anemia. Female, 43 years old, with achylic chlorosis ———December 12, 1935. erythrocytes 1.78 million, color index 0.66, sacral edema, ------December 27, 1935. after the administration of ferr. reduct., erythrocytes 3.59 millions, color index 0.55, disappearance of edema

Under certain conditions cardiac enlargement in anemia is reversible so that the heart regains its normal size and shape (fig. 137) in a short time, when the blood picture returns to normal as bone marrow function is restored or blood loss stops (Ball).

3. *The Influence of Reduced Oxygen Tension in the Respiratory Air and of Carbon Monoxide Poisoning on Cardiac Size*

Investigations on alterations of cardiac size with lowered air pressure are associated with great difficulties. Although cardiac enlargement at high altitudes has been demonstrated by percussion it is uncertain how much of this was the result of the toil associated with attaining the heights. In the Andes (4000 m.) Barcroft and coworkers demonstrated a reduction in some people investigated roentgenologically and unchanged cardiac size in others. Since, at these altitudes, cardiac rate increases considerably and this reduces diastolic dimensions (p. 131) Barcroft interpreted constancy of heart size as a sign of myocardial insufficiency resulting from hypoxia. Studies on men in low pressure chambers (LeWald and Turell) yielded no results permitting conclusive evaluation since the amount of intestinal gas, the deepening

helm, showed experimentally that copious bleeding reduced cardiac size while enlargement of the heart occurred only if the animal became anemic from persistent blood loss. This dilatation results from deficient oxygen supply and fatty degeneration of the myocardium.

Findings in human subjects are in full agreement with these experimental results After severe blood loss and copious venesection, a reduction of cardiac size has been demonstrated repeatedly, this tends to be transient (Dietlen, Assmann) since the

of leukemic myelosis and lymphadenoses, in chlorosis, in tapeworm infestations,

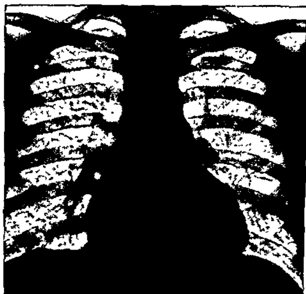


Fig 136 —Cardiac dilatation in leukemic myelosis Female, 48 years old Erythrocytes 2 88 millions, color index 0 6, leukocytes 110,000 Blood pressure 110/55 mm Hg Moderately enlarged, lively pulsating cardiac shadow with strongly pulsating curved protrusion of pulmonary arc Enlargement of both halves of the heart Pulsus celer on the aorta

and other anemias of parasitic origin, and in pernicious anemia, frequently result in enlargement of the cardiac shadow (Dietlen, Assmann, Zondek, Potter, Grunberg, as well as Tung, Bien, Chang, and others). Then, the cardiac shadow enlarges moderately to the left and right, it rests more broadly on the diaphragm and sometimes shows a flat bulge of the pulmonary arc (fig 136). Both halves of the heart participate about equally in the enlargement. Signs of pulmonary congestion are absent

Although the severity of the anemia does not parallel the degree of cardiac enlargement, the incidence of enlargement rises with the duration of the disease and the reduction of hemoglobin content.

Often the cardiovascular shadow shows very lively pulsations like those appearing from myocardial lesions They may also be assigned in part to the myocardial lesion by the anemia. In part they certainly depend upon increase of stroke volume and of the blood pressure amplitude. In favor of this opinion, Heckmann observed in

the size and shape. Often it is entirely normal even when thyrotoxicosis is rather severe (Dietlen). Very commonly, however, one can demonstrate cardiac dilatation which, at present, is considered to result partly from acceleration of the circulation and heart rate, and partly from direct action of thyroid substances on the heart muscle causing an injury which is not demonstrable microscopically for a long time.

The incidence of cardiac enlargement is a strongly debated topic. Parkinson and Cookson, as well as Misske and Schone, found an enlarged heart in about 45 per cent of their cases, Meyer-Borstel demonstrated enlargement in 83 per cent. As Holzmann correctly emphasizes, many contradictions in the literature can be resolved by the lack of adequate criteria for roentgenologic determination of cardiac size; sometimes, deviations from normal were not considered sufficiently, single cardiac measurements were employed to characterize cardiac volume, or marked protrusion of the pulmonary arc was ascribed, without further evaluation, to right heart enlargement (Meyer-Borstel). Certainly enlargement is more common with greatly increased basal metabolism and auricular fibrillation than in mild cases (Misske and Schone). Moreover, the frequency of cardiac dilatation undoubtedly increases with the duration of the disease (Parkinson and Cookson) but there is no strict parallelism between the severity and duration of thyrotoxicosis and the degree of dilatation. As previously mentioned, even in severe thyrotoxicosis, with extreme elevation of basal metabolism, marked tachycardia, and enormous weight loss, the heart may be normal in size while dilatation may appear early when signs are indefinite or definitely milder (Scherf). This variable behavior of the heart could result from constitutional and conditional factors which cause the heart to respond differently to thyrogenous substances (Bauer, Misske and Schone) Age seems to exert a certain influence since dilatation is more common, on the average, after the age of 40 (Parade, Misske and Schone). Patients who gradually become cachectic often have definitely small hearts even at advanced ages (Parkinson and Cookson), presumably their reduction of weight and blood volume with prolonged, severe thyrotoxicosis opposes cardiac enlargement (Dietlen). Also tachycardia resulting from shortening of the diastolic filling period tends to reduce cardiac volume. The coexistence of a goiter compressing the trachea, on the other hand, seems to promote the development of cardiac enlargement (Dietlen, Blauel, Muller and Schleyer, Misske and Schone).

Parkinson and Cookson compared the shape of the thyrotoxic heart to that of a ham, this comparison expresses the greater rounding and bulging of the left ventricular arc, the relatively shallow waist, and the small mid-right distance of the cardiac shadow (fig. 138) All authors stress the frequency of accentuated protrusion of the pulmonary arc which confers a more or less mitral configuration upon the heart (fig 139). According to Parkinson and Cookson, this is present in one third of the cases and according to Parade, in one half, the first figure more nearly approximates our experience. Bauer and Helm regard this as an expression of the width of the pulmonary artery, constitutionally conditioned, like the one normally occurring in childhood, in adults they regard it as a stigma of degenerative endowment upon whose soil thyrotoxicosis develops more frequently (Chvostek). Actually, relative widening of the pulmonary artery appears to be susceptible to anatomic demonstration (Parkinson and Cookson). Whether it is congenital or a consequence of thyrotoxicosis in some cases is uncertain. We often observed the conspicuous pulmonary

and acceleration of respiration, as well as fatigue and unrest of the subjects, introduced many sources of error and made the study difficult. Nevertheless, the studies of Spycher suggest that reduction of air pressure also causes cardiac dilatation. Herbst reported cardiac dilatation with a reduction of air pressure to about 260 mm. Hg (corresponding to a height of 8200 meters above sea level). He ascribed this dilatation to an increase of stroke volume, it diminished with the introduction of oxygen. Meister and Derlich obtained similar results, but they found many exceptions to this rule They attributed these deviations to variable behaviour of peripheral circulatory regulation. In particular, they found cardiac size reduced at the beginning of high altitude disease and saw it vanish with introduction of oxygen. It is highly probable that increased cardiac volume with diminishing air pressure should be regarded as myocardial dilatation from hypoxia. The regularity of this dilatation depends upon the speed with which pressure is lowered, and gradually the dilatation recedes with prolonged exposure to the same pressure (Kottenhoff). Even earlier, in animal experiments Loewy and van Leire had seen cardiac volume increase repeatedly with severe anoxemia In dogs and rabbits during asphyxia, Tullio and Businco found at first a slight reduction of heart size which soon gave way to considerable dilatation. When asphyxia was interrupted, the heart enlarged for a short time and finally returned to its original size In cats in a CO_2 atmosphere, Eppinger, Kisch, and Schwarz observed a slowly progressive cardiac dilatation which they interpreted as the result of increased influx of blood and reduced myocardial tonus.

Acute asphyxia in man also causes cardiac enlargement as observations on suffocated individuals have shown, moreover, Tullio and Businco found that cardiac size diminishes with voluntary suspension of breathing in the first 7 to 10 seconds, followed by a step-like increase of volume which is maximal in 50 to 60 seconds. This enlargement involved the right heart and sometimes also the left atrium. After ending respiratory standstill, the enlargement completely receded after a further volatile increase. In agreement with animal studies of Eppinger, Haug and Jaenisch demonstrated cardiac enlargement in man after breathing a 10 per cent CO_2-air mixture.

In illuminating gas poisoning, Zondek as well as Israelski and Lukas noted cardiac dilatation which was accompanied by tachycardia and fall of blood pressure, in a few days it vanished Whether this dilatation should be ascribed to asphyxial damage of the heart muscle, to myocardial hemorrhages which often occur, or to other degenerative events remains uncertain.

4. *The Heart in Hyperthyroidism and in Basedow's Disease*

Roentgenologic examination is important in thyrotoxicosis because the size and shape of the heart in this condition, as is well known, is often difficult to evaluate clinically. The lively pulsations which shake the anterior chest wall over a wide area tend to make the heart seem larger than it actually is (Moritz). The customary systolic murmur, the filling of the cardiac waist as demonstrated by percussion, the low grade temperature often suggest chronic rheumatic fever with mitral endocarditis. Under these conditions x-ray examination often proves helpful.

The roentgen picture of a thyrotoxic heart naturally is by no means pathognomonic or characteristic. On the contrary, it is remarkably variegated in respect to

Isolated right ventricular dilatation does not seem to play *any important role in* the enlargement of the thyrotoxic heart. Kraus, Blauel and coworkers, as well as Dietlen usually found predominant left ventricular enlargement. With postmortem control of their roentgen findings, Parkinson and Cookson usually found both chambers dilated, the left often more than the right and sometimes the left exclusively. They failed to encounter exclusive or predominant dilatation of the right ventricle. Meyer and Sulger concur in all essentials although they believe isolated right ventricular dilatation may occur.

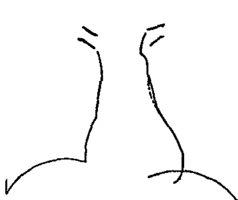


FIG. 140

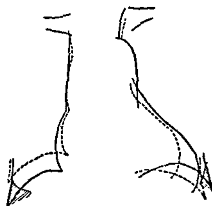


FIG 141

FIG 140—Cor thyreotoxicum Female, 33 years old. Small cardiac shadow with frequent and lively pulsations and with systolic bulging of the pulmonary arc Increased pulsations of hilar shadows. Slender aorta ——— Systolic course of pulmonary arc, - - - diastolic course of pulmonary arc

FIG 141.—Acute cardiac dilatation in Basedow's disease Female 50 years old. ------ October 4, 1935 cardiac shadow of normal size and shape with lively pulsations, EKG normal, rate 88/min, blood pressure 150/80 mm Hg, basal metabolic rate plus 59 per cent, ——— December 9, 1935, heart considerably enlarged and resting broadly on the diaphragm, pulsations arrhythmic and signs of bilateral enlargement, no signs of pulmonary congestion, small bilateral hydrothorax, EKG, auricular fibrillation, depressed S-T segment, negative T in I and II, rate 100/min, blood pressure 145 mm Hg, BMR plus 63 per cent.

In our experience the enlargement in uncomplicated cases involves both halves of the heart equally, the widening proceeds predominantly to the left and less to the right with the *heart resting broadly on the diaphragm and presenting a somewhat* aortic configuration Predominant left ventricular hypertrophy and dilatation is particularly common at advanced ages and at the climacterium, usually it can be traced to more or less marked hypertension We found predominant right ventricular hypertrophy and dilatation with coexisting emphysema or bronchial asthma As with any other myocardial injury, in thyrogenous cardiac damage, dilatation dominates in the section upon which, for special reasons, increased demands have been placed

Often the shadow suggests a relaxed heart (p 209), it rests broadly on the diaphragm, is poorly differentiated (fig 141), is remarkably labile in respect to shape

are only in the phase of systolic increase of pressure but not in the diastolic decrease (fig. 140), this suggests, perhaps, that the dilatation of the pulmonary trunk, originally dynamic, later becomes anatomically fixed.

By many authors, accentuation of the pulmonary arc has been connected with tracheal compression by the enlarged thyroid and has been called "mechanical" or "pneumatic" goiter heart. It has been suggested that tracheal stenosis imposes an overload on the right heart, it must eject more blood which streams into it as the result of the greatly reduced intrathoracic pressure in inspiration and against increasing, abnormally high intrathoracic pressure in expiration From the increased pressure in the pulmonary artery, there results right ventricular hypertrophy and





Fig. 138 Fig 139

Fig. 138 —Cor thyreotoxicum Female, 55 years old, with cardiac complaints and loss of weight for two years. Basal metabolism, plus 43 EKG auricular fibrillation, plump after-deflection Moderately enlarged, ham-shaped cardiac shadow with lively pulsations and bilateral enlargement No signs of pulmonary congestion. Aorta normal in width

Fig. 139.—Gibbus-like protrusion of pulmonary arc in Basedow's disease

dilatation (Ortner, Krehl, v. Romberg) and, accordingly, an enlarged heart with mitral configuration. New experimental studies (Sulger) suggest that tracheal stenosis does not, on the whole, produce any noteworthy increase of pressure in the pulmonary artery. Direct measurements of pressure in this vessel in tracheal stenosis, which would be important in clarifying the question, apparently do not exist. At all events, no parallelism is demonstrable between the degree of tracheal stenosis and cardiac size and shape (v. Muller, Blauel, Steiner, Meyer-Borstel, Parkinson and Cookson, Sulgar, Misske and Schone). Extreme tracheal stenosis may exist for a long time without cardiac enlargement or signs of right ventricular hypertrophy Nevertheless, with high grade tracheal stenosis, relatively often the cardiac shadow is universally enlarged and both halves of the heart are dilated (Blauel, Muller, and Schleyer, Misske and Schone, Zdansky). Moreover, thyroidectomy in these cases may cause a prompt reversal of cardiac enlargement to normal.

Misske and Schone found no parallelism between the width of the superior vena cava shadow and the height of the venous pressure. We are not inclined to place much weight upon this reported caval widening since the width is otherwise subject to extreme individual variations.

The thyrotoxic heart with its more or less mitral configuration and lively pulsations may closely resemble one with a mitral lesion in the stage of active endomyocarditis *But analysis reveals, in distinction from a mitral valve lesion,* that the left atrium is not enlarged. It is important to ascertain this, since the clinical differentiation of these diseases often encounters difficulties when the same murmurs, tachycardia, and increased temperature are present. While the left atrium of a thyrotoxic

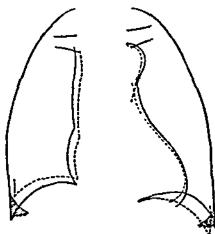


Fig 142 —Beginning cardiac decompensation in a woman, 55 years old, ill from Basedow's disease for two and one-half years. ------March 2, 1935 cardiac shadow of normal size and shape with lively pulsations, rate 110/min, blood pressure 145/0 mm Hg, basal metabolism plus 35 per cent, ———Oct 11, 1935: with increase of metabolism to plus 72 per cent the cardiac shadow has become larger, the superior vena caval shadow now wider, no signs of pulmonary passive congestion but bilateral small pleural effusions have appeared, rate 100/min, blood pressure 110/0 mm Hg

heart may dilate in the stage of decompensation this usually remains within modest limits although an absolute differentiation from mitral disease may be impossible.

Cardiac dilatation from thyrotoxicosis may regress. After spontaneous recovery and particularly after thyroidectomy it is not rare for signs of failure to recede and even for a complete return to normal to occur (Kraus, Dietlen, Blauel, Muller and Schleyer, Meyer-Borstel, Meyer and Sulger), this may happen even a few weeks after operation (Parade and Rahm). Naturally the incidence of postoperative reduction of cardiac size is estimated very differently since great differences in material are involved. In our experience, dilatations which develop in a short time, more frequently and completely receded than those appearing over the course of years; usually the latter are irreversible.

Parade and Rahm occasionally noted increased cardiac size after successful operation both in patients with hearts previously normal in size and in those with enlarged hearts which had shown a definite tendency to become smaller in the early

and size from changes in intrathoracic pressure and the position of the diaphragm (Vaquez and Bordet, Dietlen).

Usually pulsations of the cardiovascular shadow are quick and often arrhythmic owing to extrasystoles or auricular fibrillation. Often their flapping, "fickle" character (Dietlen) is striking, they correspond to Dietlen's "excited action" whose peculiarity is the speed of the excursion, particularly of the diastole phase. The breadth of the pulsation is increased in most cases (Blauel, Muller and Schleyer). This increased amplitude and flapping character of the pulsations is produced on one hand by increased systolic contraction and, on the other, by the reduction of diastolic myocardial tension (tonus) Some features of the ventricular kymogram described by Stumpf seem to support this. He observed a "flail notch" coinciding with the end of diastole and the beginning of systole which he designated an "effect of tension." Earlier, Zdansky and Ellinger as well as Westermark described a rising of the kymographic curve of the left ventricle in the tension phase as a normal feature. We believe that this can be produced by the systolic bulge and rotation of the heart (Braun) and can easily conceive that increased contractions of the thyrotoxic heart augment the bulge and rotation, thereby leading to a steeper increase of the ventricular curve in the tension period Another peculiarity of thyrotoxic cardiac pulsations is an increase of the small S-S notches located at the beginning of diastole (p. 55) if a small goiter and a large blood pressure amplitude are present. Stumpf considers this a mechanical result of the goiter We incline to the view that it may express diminished diastolic tonus (tension) of the myocardium which results in greater ventricular stretching on closure of the semilunar valves.

The higher the rate and the larger the heart is, in general, the smaller the pulsations become, this amply explains apparently contradictory reports on the breadth of the excursions along the borders of the cardiac shadow

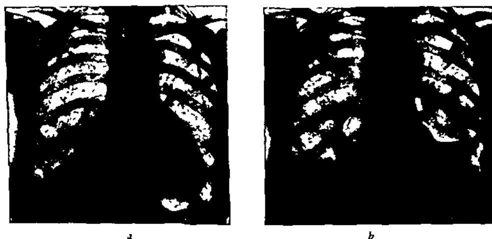
Not only the cardiac shadow, but the aorta, the trunk of the pulmonary artery, and hilar shadows as well, show lively pulsations reflecting the greater blood pressure amplitude. Reduced vascular tonus favors pulsations like pulsus celer in the aorta (Zdansky) and occasionally results in dynamic dilatation (Bayley) (p. 371) Ultimately this dilatation becomes fixed, and in conjunction with dilatation of the conus aorticus may lead to relative insufficiency of the aortic valves and its results (Luger, Wiechmann).

As a rule, the lung fields are clear in thyrotoxicosis (Rosler) since even with marked cardiac dilatation, usually pulmonary congestion fails to appear, moreover, these individuals are usually thin. The absence of pulmonary congestion in thyrotoxic cardiac failure depends upon simultaneous and equal damage to both halves of the heart. Consequently, stasis occurs exclusively or predominantly in front of the right heart. Frequently pleural effusions are associated with it. Under these circumstances the heart may closely resemble one decompensated from a combined valvular lesion

Retrograde stasis can also widen the shadow of the superior vena cava (fig 142) This expansion is mentioned by some authors (Dietlen, Rosler) as common even without signs of right heart failure. Dietlen thinks a retrosternal lobe of the thyroid or an abnormal, persistent thymus responsible. Elevation of venous pressure from dilatation of peripheral blood vessels or increased circulating blood volume have been suspected. In patients without signs of right heart failure or retrosternal goiter,

fact, thyroid preparations in themselves produce a profuse diuresis (Eppinger) which in myxedema may specifically cause collections of fluid in serous cavities to disappear.

These facts make it imperative to exercise special care in interpreting alterations of the
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a b

Fig 143 —Pericardial effusion in myxedema Female, 59 years old. (*a*) Moderately enlarged, weakly pulsating cardiac shadow widened to left and to right, on the basis of these findings, "myxedema heart" was assumed (*b*) Hydropneumopericardium after paracentesis and injection of air into pericardial cavity, the heart is normal in size

and to the absorption of a stasis hydropericardium, on the other hand, a developing myxedema can exert a cardiac-enlarging effect without permitting a decision as to which of these factors are active in a given case.

6. *Beriberi Heart*

Reinhard as well as Aalsmeer and Wenckebach described the picture of beriberi heart in detail. In the full-blown case the heart has a mitral configuration and its waist is filled more or less completely by the pulmonary conus and trunk of the pulmonary artery, both of which are dilated. In Shôshin, the terminal stage of beriberi, the cardiac shadow may reach an enormous size, there is a massive gibbus-like protrusion of the conus pulmonalis and pulmonary artery and the heart is dilated more to the left than to the right (fig. 144). All signs of right heart enlargement (Wenckebach) are found as in a mitral lesion The superior vena cava is widened considerably Numerous post mortem studies (Aalsmeer, Wenckebach, Tull) have

weeks after operation. This enlargement coincided with increase of body weight and seemed causally related to it. Moreover, the slowing of heart rate may play a role in the larger volume.

5. Myxedema Heart

Zondek was the first to call attention to enlargement of the cardiac shadow in myxedema; soon, Assmann and Meissner made the same observation. These authors ascribed the enlargement to cardiac dilatation. This conception was plausible because clinically it was clear that myxedema could damage the heart and circulation, moreover, the authors were able to show that the administration of thyroid caused the enlargement to disappear promptly.

The roentgenogram shows a moderate but sometimes enormously enlarged heart, widened to left and right, or sometimes exclusively to the left; in principal, the configuration seems to be aortic although both borders are more rounded. Analysis of the shadow seems to indicate that all parts of the heart are equally affected. Usually visible pulsations are small and sluggish, the lung fields are clear, and signs of congestion are absent Often a uni- or bilateral hydrothorax is present. Zondek also observed dilatation of the aorta which he ascribed to myxedematous injury of its wall

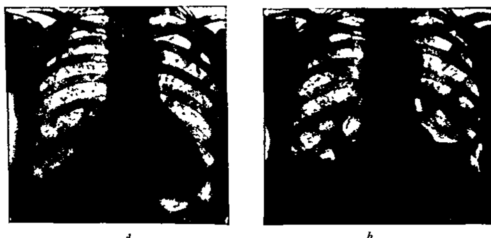
Subsequently many workers (Fahr, Zins and Rosler, E. Holzmann, Davis, Tung, Ayman, Rosenblum and Mark, Falcon-Lasses) made similar observations and estimated the incidence differently. Osler considered enlargement not unusual. Ohler and Abramson found it in seven of thirteen cases of myxedema. Other workers noted it infrequently Vaquez, Mackenzie, and Willius and Haines, among others, do not mention it.

Most authors also agree that the administration of thyroid causes the enlargement to disappear along with other symptoms of myxedema We have seen this many times. Only Meissner failed to observe a reduction in a case, presumably owing to the coexisting hypertension. After thyroid is discontinued, cardiac enlargement tends to recur in a short time (Fahr, Davis, Christian). The administration of digitalis and rest in bed exerts no influence on cardiac size (Assmann, Fahr, Davis, Christian). This evidence indicated that enlargement of the cardiac shadow resulted from hypothyroidism, and cardiac dilatation from a specific myxedematous injury of the myocardium seemed likely. Gordon and Freeman, however, noted that pericardial effusions appear in myxedema so that one must consider whether enlargement of the cardiac shadow, at least in some cases, is not produced—or not exclusively—by the heart itself but by a hydropericardium. Hurxthal confirmed this. In a

Such cases give food for thought and have made earlier interpretations of the myxedema heart problematic Cardiac damage by myxedema should, by no means, be denied. Nevertheless, one must realize that cardiac enlargement, at least in many cases, is produced by an unrecognized hydropericardium (Kaunitz, Zdansky, and Scherf). Then, the prompt reduction of the cardiac shadow in myxedema after the administration of thyroid is thoroughly comprehensible; entirely apart from this

fact, thyroid preparations in themselves produce a profuse diuresis (Eppinger) which in myxedema may specifically cause collections of fluid in serous cavities to disappear.

These facts make it imperative to exercise special care in interpreting alterations of the cardiac shadow after total or subtotal thyroidectomy has been performed (Blumgart and coworkers, Scherf, Singer and Mandl) for severe cardiac decompensation, otherwise refractory to all therapy. These authors, as well as Zdansky, repeatedly observed cardiac enlargement with decline of the basal metabolism or even before, although the circulatory situation had improved. In only a few cases did the cardiac shadow remain fixed or decrease in size (Davis, Weinstein, Risemann and Blumgart). As so often, the most diverse factors work in opposite ways. The improvement of circulation after thyroidectomy can lead to a reduction of cardiac size



a b

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Fig. 144 —Beri-beri heart in "Shôshin," the terminal stage of the disease. The heart is dilated mainly to the left and from its waist the conus and the pulmonary artery (x) protrude like a gibbus (From Wenckebach, *Das Beriberiherz.*)



Fig. 145 —Beri-beri heart, fixed in situ. Enormous dilatation of inflowing veins as well as enlargement of congested liver The anterior cardiac wall is formed mainly by the massive, dilated right heart The conus and pulmonary artery protrude from the cardiac waist (From Wenckebach, *Das Beriberiherz*)

revealed an actual dilatation of the right atrium and ventricle (often, the latter is enormous) in length and breadth with their results rotation of the heart to the left and superimposition of the left atrium by the conus pulmonalis (fig. 145) The latter, including the pulmonary valve ring and the trunk of the pulmonary artery, suffers a particularly marked dilatation so that often a relative pulmonary regurgitation is added to the coexisting tricuspid regurgitation. The left atrium and ventricle, on the contrary, are normal although hilar shadows are often enlarged (Reinhard). A pericardial effusion may contribute to the enlargement of the cardiac shadow (Reinhard, Wenckebach).

All these changes, including the pericardial effusion, can develop rapidly and disappear just as fast after vitamin therapy (Wenckebach, Hashimoto). Wenckebach regarded the changes as results of a primary damage of the myocardium and of the entire circulation which ultimately must lead to right heart failure.

7 *The Heart in Glycogen Storage Disease*

Glycogen storage disease, first described by v Gierke in 1929, is a congenital disturbance of glycogen metabolism. Apparently it is always fatal in the first months of life A massive deposit of glycogen occurs in the parenchymatous organs and in the myocardium Sometimes the myocardium may remain free. If affected, the muscle fibers are filled with clumps of glycogen separating the fibrils and distending the entire fiber (Putschar, Schneider) In this way the ventricular and atrial walls thicken considerably and the cardiac chambers may dilate. Bischoff, as well as Antopol, Heilbrunn, and Tuchmann, saw enormously enlarged globular cardiac shadows in two nurslings. In some cases, however, no cardiac enlargement occurs despite glycogen storage. It is conjectured that at times "idiopathic cardiac hypertrophy" may involve a glycogen storage in the heart muscle (H. Roesler).

IV. Coronary Sclerosis

Coronary sclerosis need not provoke any roentgenologic changes. Even when signs of coronary insufficiency are most severe and electrocardiographic changes are extreme, the roentgenogram may be completely normal Consequently, normal radiographic findings do not exclude coronary sclerosis If this sclerosis occurs in a hypertensive heart, as often happens, usually the left ventricle progressively dilates (p 206) The large cardiac shadow, widened predominantly or exclusively to the left, and its elongated, markedly rounded left ventricular arc extend far into the left lung field Signs of stasis in the left atrium and the lungs may still be absent, although often these patients complain about attacks of nocturnal dyspnea, Cheyne-Stokes breathing, and exertional dyspnea. Very gradually, or sometimes immediately in conjunction with infarction, signs of left heart failure appear, the well known picture of a mitralized aortic heart (p 187) and more or less definite pulmonary stasis develops. Thereby, the cardiac shadow may become enormous (cor bovinum). Left ventricular hypertrophy and dilatation, unequivocally demonstrated by x-ray, permits one to infer with a probability bordering on certainty, despite a normal blood

pressure, that hypertension was formerly present and consequently severe myocardial damage now exists. This is not intended to imply that every aortic heart with left-sided decompensation in a hypertensive individual absolutely proves the existence of coronary sclerosis.

Direct radiographic proof of coronary sclerosis in the living subject is possible only when large amounts of calcium have been deposited in the vessel walls, Lenk saw this for the first time in the living after Simmonds had demonstrated it in a cadaver heart. Direct proof of calcareous deposits in the vessels has little practical importance in the diagnosis of coronary sclerosis; usually calcification is missed precisely in atheromatosis of the coronary arteries and is found rather often in medial sclerosis where it lacks significance and very rarely leads to thrombosis. Kuhlmann, Wosika and Sosman, and Snellen and Nauta recognized calcified coronary vessels fairly often with good adaptation of the eyes, hard rays, and a narrow shutter.

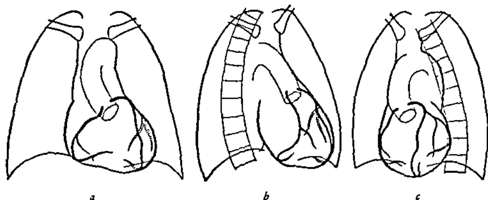


FIG 146.—Schematic representation of the large branches of the coronary arteries (*a*) Posteroanterior, (*b*) right anterior oblique, (*c*) left anterior oblique position

With short exposures they are often seen on the films as tortuous, curved or straight, double contoured streaks, nodular here and there, they take different courses depending upon the plane of projection (figs. 146 and 147) Occasionally one sees a circular shadow when the vessel is caught on-end. The form and location corresponding to the course of the vessel distinguishes them from calcification of the cardiac skeleton, the valves, myocardium, pericardium, costal pleura, or lungs.

Naturally the calcareous deposit in the vessel moves with pulsations of the heart wall. The breadth and direction of the pulsation varies according to the plane of projection (Snellen and Nauta).

It is very rare for an aneurysm of a coronary artery or an intra- or extramural hematoma to develop on the basis of vascular sclerosis (Packard and Wechsler). In the roentgenogram these aneurysms produce a protrusion of the cardiac shadow and a systolic-expansile pulsation may be seen when no thrombosis exists (Ellinger). Differentiation from an encapsulated pericardial or mediastinal effusion, as a rule, is hardly possible from the roentgenogram alone

In a cadaver heart, Jones demonstrated beautifully the normal course, ramifications, and anastomoses as well as narrowing and occlusion of the coronary vessels by injecting a contrast agent containing barium.



Fig 147.—Calcified coronary arteries. (a) anterior view, (b) right anterior oblique, (c) left anterior oblique position.

The visible pulsations of the cardiac shadow show no characteristic behavior in coronary sclerosis. If the heart is large, the pulsations are usually small. Arrhythmic pulsations are common Scherf and Zdansky succeeded for the first time in recording pulsus alternans kymographically in coronary sclerosis, the change from large to small cardiac contractions was clear. According to Stumpf, the excursions in the

cephalic parts of the left ventricular arc are usually greater than in the caudal and he

can reach remarkable dimensions, particularly in hypertensive hearts; generally speaking, they are irreversible. Usually, if the size diminishes it depends upon the absorption of a pericardial effusion (exudate or transudate).

Occasionally, x-ray examination discloses a central pulmonary edema in coronary sclerosis without cardiac decompensation

V. Coronary Occlusion

Coronary occlusion may result from embolism, aortic lues, or thrombosis. Embolic occlusion of a coronary artery is rather rare while extreme narrowing or

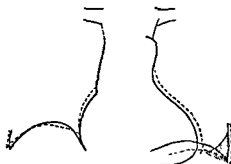


FIG. 148 — Left sided pleural effusion after myocardial infarction Male, 34 years old. ———three days after infarction, ------twelve days later the pleural effusion has vanished The cardiac shadow has become somewhat larger

complete occlusion of a coronary ostium by syphilitic aortitis is a common event Since coronary ostial stenosis tends to develop very slowly, collaterals often take over the nutrition of the myocardium so that no stormy manifestations or cardiac alterations, demonstrable roentgenologically, need occur, rather, these coronary occlusions are often first discovered at necropsy. Only when severe nutritional myocardial disturbances occur, but not always even then, the heart may dilate but this becomes considerable only when aortic valve regurgitation or hypertension coexists

The most common and important type of coronary occlusion is thrombosis which ordinarily develops on the basis of sclerosis of its wall Naturally, coronary thrombosis as such is not detectable by x-ray. Nevertheless, owing to myocardial damage, cardiac failure may progress so that the heart enlarges acutely (fig. 148). Apparently these enlargements are reversible. Occasionally cardiac dilatation may be simulated by a pericardial effusion (Schwartz), but this is uncommon because post-infarction pericarditis usually does not create a large effusion. In one of our cases, hemopericardium from rupture of an infarcted left ventricle acutely enlarged the cardiac shadow.

None of these *manifestations need be present so that the cardiac shadow may be* normal in size and shape despite extensive infarction

Occasionally, the pericarditis extends to the left pleura and produces a left pleural effusion (fig. 148).

In some left-sided coronary occlusions, Borak observed elevation and limited shift of the left diaphragm. He thought that extension of the pericarditis to the diaphragm caused a functional disturbance. Recently, Laubry, Soulié, and Heim de Balsac confirmed this observation and assigned some diagnostic significance to it. They also saw a left-sided diaphragmatic paresis which they ascribed to phrenic nerve injury by pleuro-pericardial scars forming after a myocardial infarction. According to them, the elevation of the left diaphragm occasionally results from increased gas in the stomach and splenic flexure. Certainly, immobility of the left diaphragm from reflex causes after myocardial infarction could favor an abnormal collection of gas.

Acute and subacute pulmonary edema frequently appear in conjunction with coronary thrombosis but are rarely seen by roentgenologists. As a rule pulmonary congestion is missed when the thrombosis occurs in a heart previously normal in size and working under normal conditions. But, if the left coronary artery of a hypertensive heart is involved, as frequently happens, with progressive dilatation of the left heart, signs of mitralization and pulmonary congestion appear.

The focus of myomalacia and the subsequent scar usually are not demonstrable by x-ray. Even a retracted scar on the cardiac surface escapes detection. Nevertheless, roentgenkymography succeeds in locating the softened area in about 75 per cent of the cases (Stumpf, v. Braunbehrens, Schilling, Arendt, Bickenbach, Cramer and Stehr, Sussmann, Dack and Master, Heim de Balsac and coworkers). By means of electrokymography these results were improved in several respects (Luisada and Fleischner, Heckmann, and others). The leading signs are: (1) reduction or complete absence of pulsatory excursions and (2) inverse (systolic-expansile) pulsations in a circumscribed area of the ventricular surface. The absence of pulsations in a circumscribed place characterizes the "mute zones" and are designated by Luisada and Fleischner as a "local paralysis." The inverse pulsations can be found in the region of a protruding area when a cardiac aneurysm (p. 234) exists, or may even appear without such a bulge, in the latter instance, Zdansky and Luisada and Fleischner speak of a "dyamic aneurysm" (p. 230). Apart from these significant signs, slight alterations are found in the ventricular kymogram especially in the vicinity of myomalacia and cicatricial foci, these consist, for example, of a premature end and belated start or deformation of the systolic limb of the curve and an abnormally high rise or deformation of the diastolic limb of the curve (Luisada and Fleischner) Usually these alterations do not appear until the end of the first or second week although occasionally Master saw systolic lateral pulsations immediately after the occlusion. Sometimes roentgenkymograms are positive in patients whose electrocardiograms are normal so that these diagnostic methods are supplementary. Usually roentgenkymographic findings remain unchanged for months and years. The findings may, however, improve or become worse as the pathologic process progresses and may have prognostic value.

Occasionally in the region of a myocardial cicatrix, shadows are produced by

calcification of parietal thrombi or by calcification or ossification of the cicatrix itself (Scholz). Clark observed calcification of a cicatrix over a period of ten years.

VI. Cardiac Aneurysm

By cardiac aneurysm* one understands a circumscribed bulge of a cardiac cavity where the wall becomes thin following a myocardial injury. This acquired bulge merely represents an indentation of the inner surface of the cavity affected, or it may be a protrusion on the cardiac surface.

There are acute and chronic cardiac aneurysms.

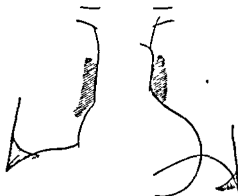


FIG 149.—Extensive myomalacia affecting almost the entire left ventricle and ventricular

An acute aneurysm develops on the basis of an ulcerative mural endocarditis or a purulent myocarditis, it plays no practical role in roentgen diagnosis since these patients are too ill for detailed roentgen examination

Chronic aneurysms alone come under consideration. They develop from a stab or gunshot wound, more rarely from gumma, and most commonly from focal myomalacia after coronary occlusion. In rare cases they seem to develop very acutely, Shookkoff and Douglas described an aneurysm demonstrable by x-ray six days after the infarction. Spherical distention of an extensive infarct of the left ventricle and apex, which we described in a hypertensive patient (fig. 149) who first became ill on the day prior to x-ray study, may, perhaps, be conceived as abnormal stretching of the wall which was beginning to yield, that is, an acute dynamic aneurysm of the cardiac wall. Aubertin and Horeau observed an aneurysm of the left ventricle six weeks after infarction

The statement of Groedel that the diagnosis of these aneurysms cannot be made without x-ray is not valid as a generalization (Scherf and Erlsbacher, Dressler) although roentgen study actually performs valuable service in their recognition. Certainly, only a minority of cardiac aneurysms can be detected by roentgenograms.

* Excessive dilatation of the atrium, often called atrial aneurysm, is excluded here.

This is explained partly by the fact that many of them involve only the inner layers of the wall or are so circumscribed and so shallow that they gradually blend with the surrounding areas. Primarily, however, roentgen discovery of these aneurysms is difficult because of their unfavorable location. As is well known, most of them are located at the apex and consequently project into the abdominal shadow from which they cannot be demarcated even when large (Dietlen, Vogt). Aneurysms of the posterior wall of the left ventricle and of the anterior wall of the right can be detected only in the left anterior oblique position (Schwedel and Gross). Aneurysms of the ventricular septum almost always escape detection. Only isolated examples have been described (Bauke, Wachner and Brenner, Boller and Pape). Aneurysms discovered roentgenologically up to the present and confirmed by necropsy or other



FIG. 150 —Aneurysm of the left ventricle in a hypertensive patient, 60 years old, four months after a myocardial infarction The bulging aneurysmal shadow shows no pulsations. Pulmonary congestion indicates failure of the left ventricle

clinical findings, almost without exception presented a circumscribed or a more diffuse bulge corresponding to their most common site, this area is supplied by the descending branch of the left coronary artery and usually the aneurysm involves the left cardiac border As a rule, they are near the atrioventricular junction in the middle of the left ventricular arc (fig. 150) or near the apex (fig 151) (Kraus, Bergonié and Moutner, Sézary and Alibert, Heitz and Corone, Jaksch-Wartenhorst, Lenk, Kalisch, Golonsko, Melchart, Wiberg, Zadek, Groedel, Kuhlmann, Parade, Steel, Regelsberger, Schwedel and Gross) Apical aneurysms often are not sharply outlined against the rest of the chamber, part of the ventricle dilates and its cavity blends with the aneurysm (Monckeberg). For this reason it often resembles a simple elongation of the left ventricle Only when the aneurysm has attained a fair size (fig. 152) is there a circumscribed protrusion of the apical section, Zadek calls this "diffuse eccentric rounding of the apex." The resultant shape of the heart, as Zadek concedes, often cannot be definitely differentiated from aortic valve



FIG 151 —Large aneurysm of the apex, six months after myocardial infarction in a man, 41 years old Small ventricular pulsations were present on the aneurysmal shadow

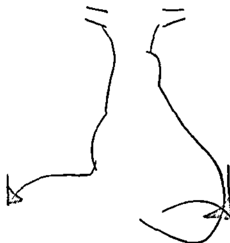


FIG 152.—Large aneurysm of the cardiac apex. Hypertensive patient, 60 years old, four months after myocardial infarction The aneurysmal shadow shows systolic-expansile pulsations alternating with those in parts of the left border near the base. The aneurysm produced an indentation of the gastric gas bubble Bilateral small hydrothorax.

regurgitation or a decompensated hypertensive heart. When one recalls how often coronary sclerosis and hypertension are associated, the presence of an apical aneurysm will be assumed only with great reserve on the basis of this shape. The fact that Zadek assumed an apical aneurysm to be present in 50 per cent of all cases of coronary sclerosis on the basis of "diffuse eccentric rounding of the apex" is a

warning in this respect. Probably a majority of them formerly had hypertension but the blood pressure now had fallen owing to myocardial damage.

It is not always possible to differentiate an extensive aneurysmal bulge of the anterior wall of the left ventricle from a dilatation (Schwedel and Gross). If, of course, the entire left border of the heart or only one part is distended like a balloon so that it bulges at an angle below the cardiac waist (Bauke, Parade, Melchart, Zdansky) *(fig. 153) one may be almost certain that an aneurysm of the left ventricle* is present.



Fig. 153 —Large, balloon-like aneurysm protruding from the left ventricle two months after myocardial infarction Male, 43 years old The aneurysm shows systolic-expansile pulsations.

Usually the aneurysmal shadow is sharply defined, simply arched, slightly protruding or angular, sometimes it is vaguely defined and shows notches or irregularities extending toward the lung fields, the diaphragm, or anterior chest wall. This indicates adhesions resulting from postinfarction pericarditis. In this connection it *may be recalled that a left pleural effusion after myocardial infaction* may leave pleural adhesions behind

As a rule, the aneurysmal shadow is devoid of structure Rarely, calcareous deposits are seen, as Simmonds first demonstrated in the roentgenogram of a cadaver heart. Jaksch-Wartenhorst described a spherical deposit, about the size of a walnut, showing lively movement with cardiac action, which corresponded to a calcified mural thrombus. Sézary and Alibert saw a dense shadow which they interpreted as mural calcification or calcified masses of thrombus Once, Kuhlmann recognized a calcified *afferent vessel* Mural calcification of a cardiac aneurysm, confirmed by

necropsy, was reported by Brenner and Wachner. This case is noteworthy because of the location The roentgenogram showed a round structure, the size of an apple, projecting to the left from the cardiac waist and practically outlined by a calcareous shell As necropsy showed, a chronic saccular aneurysm with thin walls emerged from the posterior wall of the left ventricle near the base of the heart to which it was connected by an opening 25 mm. wide It was filled by masses of thrombin and the wall was calcified Heim de Balsac and Marquis also observed, ten years after infarction, a calcified cardiac aneurysm, the size of a pigeon egg, with a calcified edge projecting from the left border of the heart and showing systolic expansile pulsations Schwedel and Gross mention three cases with calcification demonstrable roentgenologically Occasionally a calcified deposit facilitates the discovery of a cardiac aneurysm which does not project outward but has merely thinned the wall, this is presumed to have happened when the calcification is located at a distance *from the cardiac surface which is less than the mural thickness of the affected section* of the heart

Since cardiac aneurysms consist of scar tissue, contain no muscular elements capable of contraction, and are often filled with massive thrombi, their surfaces may show only small pulsations or complete immobility. Not rarely, the bulge of an aneurysm shows systolic expansile pulsations when the systolic increase of pressure passively distends its thin wall These pulsations are usually striking because they alternate with those of the neighboring parts of the ventricle (fig. 153) Sometimes these pulsations are distinct only in the right lateral position

Occasionally a systolic retraction is observed on the border of the aneurysmal bulge (Assmann) Finally, Christian and Frik saw lateral systolic pulsations along the edge of an aneurysm, they were produced by the systolic contraction with rolling and rounding of the surrounding intact ventricular muscle The pulsations just mentioned have been described in roentgenkymograms by Stumpf, Heckmann, Cramer and Stehr, and others Heckmann thought they always merged gradually with the pulsations in the adjoining areas of intact muscle. Cramer and Stehr as well as Aubertin and Horeau also showed dual notch formation in the region of the aneurysm

Many years ago, Groedel demonstrated systolic expansile pulsations by roentgen cinematography

In exceptional cases, the radiographic findings together with the clinical manifestations permit one to assume the presence of an aneurysm even when it does not reach a cardiac border Thus, a patient had luetic aortic valve insufficiency, hypertension, *right hydrothorax, congested liver and edema (Boller and Pape)* The cardiac shadow was decidedly enlarged, widened particularly to the right, and rested broadly like a pear on the diaphragm, the left ventricular arc, however, showed no elongation or marked rounding which would have been expected from a decompensated aortic valve regurgitation Since the left atrium was not enlarged and pulmonary stasis was absent, since no signs of a congenital anomaly or pulmonary valve lesion were present, *the authors concluded that an aneurysmal dilatation of* part of the heart existed. Actually, at necropsy, a cup-shaped aneurysm of the lower half of the interventricular septum was found, it bulged toward the right ventricle, narrowed its lumen and displaced the right heart wall to the right.

Traumatic aneurysms after stab or gunshot wounds of the heart are rare and not essentially different from those developing after infarction. Kienbock described a small aneurysm of the posterior wall of the left ventricle where an infantry bullet had lodged. Bianchi interpreted a pulsating shadow projecting from the cardiac waist after a shrapnel wound as a traumatic aneurysm at the base of the left ventricle In a woman who had shot herself in an attempt at suicide, we saw a date-sized, nonpulsating angular protuberance on a somewhat smaller base resting on the left ventricular arc just above the apex (fig. 154) In this instance we postulated an aneurysm of the left ventricle.

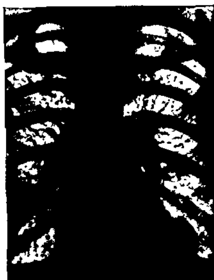


Fig. 154 —Aneurysm of the cardiac apex(?) Gunshot wound of the heart three years earlier. Female, 31 years old.

In summary, the roentgen diagnosis of aneurysms of the cardiac wall is relatively infrequent This depends upon the prevalence of small aneurysms and a position usually unfavorable for demonstration by x-ray In a rather unusual location at or near the base of the ventricle a cardiac aneurysm is relatively more accessible from a roentgenologic standpoint than the ordinary apical lesion Moreover, a differential diagnosis is rendered more difficult because a variety of lesions may create similar roentgen pictures.

1. Tumors and tumor-like structures in the mediastinum, the root of the lung, pericardium, or the heart. It is rather exceptional for these to show systolic expansile pulsations (very vascular sarcoma, hemangioma, metastasis from hypernephroma) The presence of signs of a mediastinal or pulmonary tumor as well as diaphragmatic paralysis speaks against cardiac aneurysm. Benhamou described an echinococcus cyst of the left heart which closely resembled a cardiac aneurysm.

2 Small, encapsulated pleuromediastinal effusions lying on the heart. In this instance a diminution or disappearance of the shadow alone permits a differentiation.

3. Aneurysm of the sinus of Valsalva (p. 392) causes a pulsating shadow sometimes with a calcium deposit at its border, usually it extends to the right and forward and therefore the position distinguishes it from a cardiac aneurysm.

4. Aneurysms and intra- or extramural hematomas of the coronary arteries (Ellinger) may develop on an arteriosclerotic, mycotic, or luetic basis (Packard and Wechsler), they are extremely rare.

5. Encapsulated pericardial effusion ("inflammatory pericardial diverticulum") and true pericardial diverticulum Both produce differentiated, well defined soft tissue shadows resting broadly on the heart. Encapsulated pericardial effusions are usually located along the right cardiac border while cardiac aneurysm is practically always found on the left. The sole right-sided cardiac aneurysm described in roentgenologic literature (Kirschmann) was not established by necropsy.

6 On deep inspiration the "fat pad" may project as a laterally convex shadow [illegible]

[illegible]

earlier myocardial infarction, one may easily be induced to consider an apical aneurysm. A definite decision is not always possible (Zdansky)

VII. Cor Pulmonale

Cor pulmonale develops when the right ventricle is saddled with excessive work owing to primary changes in the pulmonary circulation or from abnormal respiratory mechanics

Accordingly, all conditions which narrow the pulmonary circulatory bed, such as emphysema or chronic indurative and cirrhotic-cavernous pulmonary tuberculosis associated with widespread destruction of the parenchyma, pneumoconiosis and other nonspecific indurative processes, primary pulmonary sclerosis, thromboangiitis obliterans of the pulmonary vessels, and pulmonary embolism, come under consideration as causes of cor pulmonale, furthermore conditions which dynamically throttle pulmonary circulation, like bronchial asthma, severe chronic bronchitis, kyphoscoliosis, pleural adhesions, extreme tracheal stenosis, and finally certain occupations (glass blowers) requiring a marked increase of intrapulmonary pressure, may be provocative

Roentgenologic examination of these hearts has greatest practical importance since in all of these conditions cardiac size is mistakenly judged on physical examination, usually the size is underestimated but occasionally it is overestimated because the heart is covered by emphysematous lungs or cannot be unequivocally examined owing to dullness created by pleural scars, pulmonary induration, or thoracic deformity

Cor pulmonale is classified as acute, subacute, and chronic. The roentgenogram of acute cor pulmonale resulting from pulmonary embolism has not been described up to the present. Subacute cor pulmonale often develops from narrowing of the pulmonary vascular stream by tumor emboli or blood clots, and compression or thrombosis of the small pulmonary vessels in lymphangitis tumorosa, as well as actual invasion of small lung vessels. The heart is enlarged only a little and shows a

more or less distinct mitral configuration owing to the prominence of the pulmonary arc (Brill and Robertson, Mason). We had occasion to observe the development of subacute cor pulmonale. A 27 year old woman with a pancreatic carcinoma suddenly collapsed a day before death (fig. 155a and b) The heart, previously normal in size and shape, then enlarged considerably and displayed a mitral configuration owing to protrusion of the pulmonary arc. Both ventricles were dilated moderately while the left atrium was normal. The superior vena caval shadow was wide. Undoubtedly an acute rise in resistance of the pulmonary circulation and pulmocoronary reflexes (Scherf) precipitated sudden cardiac failure with dilatation. In a second case*

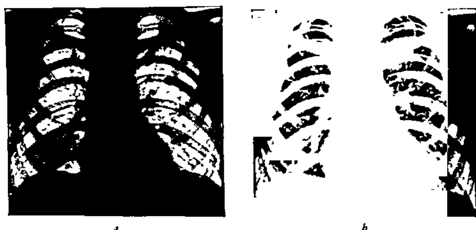


FIG 155 —Acute cor pulmonale resulting from embolic occlusion of numerous small branches of the pulmonary artery by cells of a pancreatic carcinoma (necropsy). Female, 27 years old (*a*) November 9, 1933, cardiac shadow of normal size and shape. (*b*) December 6, 1933 (one day before death), enlarged cardiac shadow with mitral configuration owing to flat bulge of pulmonary arc Both ventricles enlarged, the right more than the left Left atrium normal in size The hilar shadows were large but sharply defined Lung fields normally light. The superior vena cava has become wider. The electrocardiogram showed signs of myocardial damage

multiple pulmonary emboli from wide-spread pelvic thrombosis developed in the eighth month of pregnancy. Despite the most profound circulatory failure with fainting, tachycardia, and lowered blood pressure we found the heart normal in size with a highly elevated diaphragm and consequent mitral configuration, there were no signs of right heart dilatation. The outstanding feature was the narrow aorta whose diameter (Kreuzfuchs' method) was 2 cm This observation shows that in other cases failure can occur without dilatation of the right heart. The latter fails to appear because the influx of blood into the heart is reduced, obviously the smallness of the aorta was dynamic and at necropsy its dimensions were normal.

Chronic cor pulmonale is characterized by hypertrophy with more or less dilatation of the right ventricle. This leads to alterations of the cardiac shadow which were described in detail on page 143. Through elongation of the outflow

* The case was described in detail by Scherf and Schönbrunner to whom I am indebted for the clinical data.

tract of the right ventricle, and dilatation and lifting of the dilated conus pulmonalis and dilated pulmonary artery, as well as through rotation of the entire heart to the left, the cardiac waist is filled more or less completely, that is, a mitral configuration appears (fig 156). In primary pulmonary sclerosis accompanied by cyanosis, the pulmonary trunk often shows very striking dilatation (Arrillaga, Darley and Doan) so that the pulmonary arc protrudes from the waist like a gibbus. In some of these cases, syphilitic mural damage of the pulmonary artery is responsible (Hare and Ross)

Often cor pulmonale can be distinguished from a mitral lesion only by the absence of left atrial enlargement In a great many cases the mitral configuration is merely suggested since the conditions enumerated earlier are often accompanied by diaphragmatic descent, this opposes lifting the pulmonary conus and pulmonary artery as well as the rotation of the heart to the left (p. 175). When there is an associated hypertension, filling of the cardiac waist is usually offset by greater bulging of the left ventricular arc and the aortic knob, then a very large globular heart is often seen.

In most cases of severe pulmonary emphysema the heart is not enlarged nor has it a mitral configuration; on the contrary, it is strikingly small, median, and normal in shape. Signs of right ventricular hypertrophy are missed in the examination. Actually the pathologist also finds, in some cases of severe emphysema, no trace of hypertrophy or even dilatation of the right ventricle. This indicates that the resistance in the pulmonary circulation in pulmonary emphysema need not be high (Uhlenbruck), and intracardiac measurements of pressure have also actually shown that the systolic pressure in the right ventricle itself is not always increased even in the presence of a considerable emphysema (Cournand and coworkers). The diminutive heart in these patients often contrasts strongly with the severity of the existing cyanosis and dyspnea. This apparent contradiction is explained by the fact that respiratory incompetence stands in the foreground and cardiac failure in the background. Defective arterialization of blood depends primarily upon reduction of pulmonary respiratory surface as well as the reduced vital capacity resulting from thoracic rigidity and descent of the diaphragm On one hand, the heart must contend with heightened resistance in the emphysematous lung while, on the other, it is relieved by impaired respiration which causes stasis of blood in front of the heart (inflow stasis). Consequently, the heart is poorly filled and often very small (Sotier). Sometimes the heart enlarges in ultimis when progressive hypoxemia damages the myocardium and causes dilatation However, this may remain absent until just before death.

A small heart is found in many patients with emphysema, bronchial asthma, and pulmonary tuberculosis, in some instances this certainly depends upon a constitutional hypoplasia (Kraus, Bauer, v Hoesslin), in part, it is acquired through enforced physical inactivity and the impaired physical condition of the patient (Dietlen), to some extent, it is simulated by inadequate filling in the upright position (Sotier) as can be demonstrated by examination in the recumbent posture. Then, it is not unusual for hearts which appear small while the patient stands, to show actual enlargement This enlargement is not limited, as a rule, to the right heart but also affects the left. Nutritional disturbances of the myocardium from chronic hypoxia,

coronary sclerosis, or toxic-infectious agents are the commonest causes of these dilatations

Although the heart in emphysema, asthma, and chronic tuberculosis is usually normal in size, it may be definitely small Enlargement usually keeps within modest limits in the absence of failure. Binhold frequently found the heart enlarged but he judged cardiac size by the Kahlstorf ratios which, in underweight individuals—with which we deal very frequently—may easily simulate enlargement.

The pulsations of the cardiovascular shadow are not characteristic. With adhesions in the mediastinum or mediastinal pleura, pulsations may vanish in some places Sometimes, in marked emphysema we observed pulsations of the cardiac borders which were almost flapping as in a pneumothorax.



Fig 156



Fig 157

Fig 156 —Cor pulmonale Male, 34 years old, with severe emphysema and chronic bronchitis Bulging of pulmonary arc Enlargement of and augmented intrinsic pulsations of hilar shadows.

Fig 157 —Decompensated cor pulmonale in a young woman with bronchial asthma and severe emphysema Severe cyanosis Hepatic congestion

In typical cor pulmonale the lung fields are abnormally clear unless they are *obscured by tuberculosis, nonspecific inflammatory processes, pleural scars, or* chest deformities If this is not the case, the hilar shadows may be enlarged and the perihilar vascular markings accentuated reflecting elevated pressure in the pulmonary arteries Often the hilar shadows show large systolic expansile pulsations (Savini, Gerhartz) (p 148)

Owing to the high position of the upper chest aperture, usually present, the apical fields are rather small. For the same reason, the thyroid shadow is often retrosternal and should not be mistaken for a retrosternal goiter It is common for the thyroid to appear in the thoracic cavity when the upper chest aperture rises and its dorsoventral diameter increases.

The diaphragm is low and flat, its respiratory movements are very limited. In severe cases it shows a short, jerky inspiratory movement and a very slow, laborious expiratory rise Flattening of the domes is most distinct in lateral positions, often the

diaphragm has completely lost its curve and it descends as a flat ridge from in front and above to below and posterior. This position shows best how small the respiratory excursions of the diaphragm are and accordingly provides an impressive picture of reduced vital capacity

In obese individuals and with good tonus of the abdominal wall, the descent of the diaphragm may be slight or entirely absent. Then, the respiratory excursions of the diaphragm are less restricted.

In lateral views, the depth of the thorax is very distinct; there is accentuated bulging of the anterior wall and increased kyphosis of the spine. Moreover, the retrosternal field is widened and translucent since the distended lung is inserted between the heart and the anterior chest wall.

With failure of the right ventricle the right heart usually enlarges progressively. The resultant alterations of the cardiac shadow (fig. 157) were discussed on page 143. There it was shown that right ventricular dilatation tends to enlarge the cardiac shadow predominantly or exclusively to the left and that generally it enlarges decidedly to the right only when the left ventricle is simultaneously dilated

Descent of the diaphragm, frequently present with cor pulmonale, creates special situations so that there are many exceptions to the rule just given. The heavy heart, bereft of its normal support, seeks a median position, divided equally to left and right, irrespective of whether the enlargement involves the right half or both halves. Accordingly, even an exclusive right sided enlargement may lead to a globular heart which bulges equally to left and right (fig 158) as Vaquez and Bordet noted without explaining. The marked rounding of the left cardiac border and the protrusion backward and to the left can suggest left ventricular hypertrophy and dilatation in the anterior and left anterior oblique positions. Here, roentgen analysis of the cardiac shadow often fails completely. The absence of aortic dilatation and the presence of emphysema in such cases should raise the question that marked expansion to the left is not produced by the left heart but by enlargement of the right.

An inspiratory swelling of the cardiac shadow is often seen with emphysema and asthma, it was described by Holzknecht and Hofbauer and was confirmed by Assmann and by Moritz, Dietlen, Goetzel and Kienbock, Zdansky and Ellinger, and Nolte and Weltz investigated and analyzed it by the roentgenkymograph (p. 108).

Cor pulmonale often experiences drastic deformity from pleural and pleuromediastinal scars Especially in cirrhotic tuberculosis associated with emphysema, contraction of the pleuromediastinal scars stretches both borders of the mediastinum, thus, differentiation of the cardiac shadow into single arcs is completely lost and the cardiac shadow may resemble an equilateral triangle with its small base on the diaphragm

A deformity of the cardiac shadow which should not be ascribed to cor pulmonale is observed in contracting, left upper lobe processes and left-sided mediastinal and interlobar scars. Often, by traction these processes produce hump-like protuberances and angular kinking of the pulmonary arc (Weinberger, Dietlen) and consequently a mitral configuration (fig. 159). Occasionally in these cases a diastolic murmur of regurgitation is audible to the left of the sternum (Neumann, Scherf), it is produced by relative incompetence of the pulmonary valves resulting from distortion of the ring.

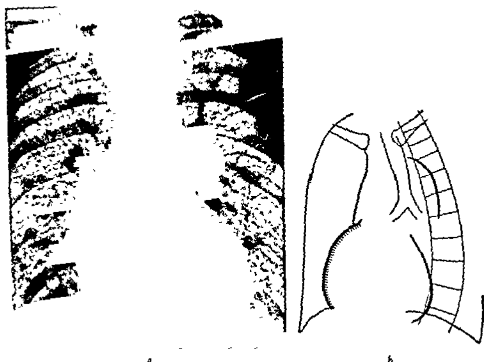


FIG 158.—Decompensated cor pulmonale with severe emphysema (necropsy) Male, 47 years old Very large cardiac shadow, widened to left and right with elongation and flat bulge of pulmonary arc Examination in the left anterior oblique position (*b*) shows that the enlargement exclusively involves the right heart Likewise the narrow aorta (arch diameter = 2 3 cm.) speaks against hypertrophy of the left ventricle Necropsy showed that the large hypertrophied right ventricle occupied almost the entire anterior wall of the heart and displaced the normal left heart backward



Fig 159 —Traction on pulmonary artery by a contracting fibrosis of the left upper lobe.

VIII. Tumors of the Heart

Primary tumors of the heart are rare Some are congenital. They include fibroma, leiomyoma, rhabdomyoma, angioma, sarcoma, myxoma and lipoma. Three fourths of the primary tumors are benign (Prichard) and half of them are myxoma. Some consider myxoma as organized masses of thrombi with mucoid metaplasia, but Prichard considers them true tumors. The atria are involved more frequently than the ventricles, the left heart more than the right. The clinical and roentgenologic signs of cardiac tumors are extremely diversified and depend upon their size and site. Corresponding to their rarity and, usually, their very nonspecific symptoms, only isolated radiographic findings of cardiac tumors are known

Direct roentgenologic demonstration of a tumor is usually impossible With a solid infiltrating sarcoma of the right atrium, Popp saw a marked nonpulsating bulge of the right cardiac border. Solitary and multiple rhabdomyoma occasionally project definitely from the cardiac surface and consequently are demonstrable by x-ray (Bradley and Maxwell) Similar shadows, however, can develop from primary and secondary neoplasms of the pericardium, of the mediastinal pleura, from encapsulated mediastinal or pericardial effusions, pericardial diverticulum, cardiac aneurysm, echinococcus cysts (Moreau and Boudin), gumma and so forth. In angiosarcoma the protrusion may show a systolic expansile pulsation, however, Rosler saw such pulsations in a liquefying gumma which openly communicated with the left ventricle. Ellinger saw them in aneurysma spurium of the right coronary artery. Diffuse rhabdomyomatosis involving much or all of the heart may lead to an aortic configuration (Pauli) or to nonspecific enlargement of the cardiac shadow, the coexistence of tuberous sclerosis makes the assumption of rhabdomyomatosis likely in some cases (Chiari) Pauli observed congenital diffuse rhabdomyomatosis of the heart in two sisters

The indirect signs of cardiac tumors are somewhat more common but naturally even more ambiguous than the direct. Intracardiac tumors like myxoma or polypoid sarcoma may enlarge single cardiac chambers by virtue of their size. Ehrenberg observed a polypoid sarcoma of the right atrium with considerable enlargement of the cardiac shadow to the right and lively pulsations of the right cardiac border. Myxoma, which is relatively common, often is pedunculated and located above the atrioventricular ostium. Therefore it may act like a valve and sometimes causes *persistent or intermittent obstruction* When located in the left atrium, apparently the favorite site, narrowing of the mitral ostium may produce all clinical and roentgenologic signs of mitral stenosis with stasis in the lungs (Ludwig, Chiari, Block and coworkers). Tumors in the conduction system may cause heart block or other conduction disturbances which may be detected roentgenologically. Extension to the cardiac surface usually results in a pericardial effusion and this may be the sole but nonspecific sign of the tumor.

Secondary tumors of the heart (sarcoma, carcinoma, chondroma) are much more common than primary ones. They enter the myocardium either through the blood stream, the lymphatics, the cardiac cavities with the blood or by continuity from the large veins, finally, a tumor may grow into the heart from surrounding organs Like primary tumors, secondary ones scarcely produce characteristic clinical or

roentgenologic signs. Occasionally the cardiac shadow may be irregular in outline Often they cause pericardial effusions or hemopericardium. We observed a woman who had undergone operation for a thyroid tumor, she developed a nodular neoplastic shadow which projected from the right upper mediastinum and simultaneously extreme stasis appeared in the domain of the superior vena cava At necropsy a recurrent tumor had ruptured into the left innominate vein and had grown down the superior vena cava into the right atrium, the tumor mass completely filled and distended the vena cava.

In some cases a diagnostic pneumopericardium or pneumothorax facilitates or makes possible the decision whether the questionable shadow belongs to the heart. the pericardium or the lungs (Roesler)

IX. Alterations of the Cardiovascular Shadow from Pleural and Pulmonary Processes

The heart suffers various displacements and deformations from space-occupying or contracting processes of the lungs and pleura.

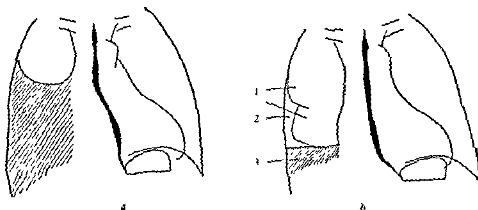


FIG 160.—Displacement and rotation of the heart to the left by a right sided pleural effusion (a) Pulmonary arc projects as a flat bulge in the cardiac waist The esophagus is displaced to the left (b) After thoracentesis the cardiac shadow regains its normal position and shape The esophagus now proceeds normally

1 Partly collapsed lung
2 Pneumothorax
3 Residual effusion

Large right pleural effusions displace and rotate the heart to the left. This accentuates the projection of the pulmonary artery into the cardiac waist and produces a more or less outspoken mitral configuration (fig 160a and b) Left pleural effusions displace the heart to the right so that the right cardiac border projects far into the right hemithorax Large pleural effusions, on the other hand, which cannot be demarcated from the cardiac borders, may make a decision about heart size difficult or impossible By filling the esophagus with barium, however, an impression can be obtained on the extent of displacement and from this, with definite

limitations, an idea can be formed on how much bulging of a cardiac border lying next to an effusion is produced by cardiac enlargement and how much displacement is produced by the effusion Likewise, dorsal displacement of the retrocardiac section of the esophagus may help to form a judgment on cardiac enlargement or pericardial effusion.

Encapsulated mediastinal pleural effusion broadly in contact with the heart often cannot be distinguished from it (fig 161) and may simulate cardiac enlargement, cardiac aneurysm, or pericardial diverticulum (p. 331). It is often difficult to

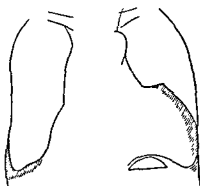


Fig. 161

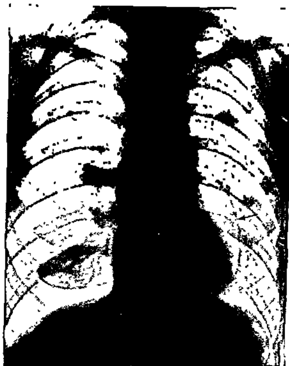


Fig 162

Fig 161 —Encapsulated left mediastinal effusion (necropsy) Female, 49 years old The mass of shadows, indistinguishable from the heart showed transmitted pulsations and was lifted in inspiration with the anterior chest wall.

Fig 162 —Overdistention of the anterior weak area (anterior mediastinal hernia) with right sided artificial pneumothorax Palpitation and pain in the cardiac area The overdistended mediastinum projects as a delicate arc over the cardiac shadow into the left lung field (arrow)

differentiate such effusions from aortic aneurysms. Pleural fluid in the anterior or posterior costomediastinal angle may produce double contour formations which usually resemble a right angle triangle whose long side is formed by the spine and whose short side is the diaphragm. The hypotenuse of this triangle naturally need not proceed as a straight line but may show a lateral concave or convex course

The heart also experiences displacements or changes of shape in pneumothorax similar to those in pleural effusions. Essentially the cardiac distortion is produced by the retraction force of the opposite expanded lung. Very striking are the large lively flapping pulsations of the cardiovascular border on the same side even with

small collections of air in the pleural cavity. The pulsations are identical with those seen on the heart of an experimental animal with an open chest the heart liberated from the elastic tug and pressure of the surrounding lung, which acts almost as a damper, in diastolic relaxation briskly distends from inrushing blood and, in consequence to its change in filling, experiences a considerable displacement, this is favored by a firm opposition of the other lung. In a left pneumothorax, Heckmann described a protrusion of the left cardiac border into the cardiodiaphragmatic angle which showed lateral systolic (paradoxic) pulsations

The extent of cardiac displacement by a pleural effusion or pneumothorax varies decidedly It depends upon the difference of pressure in the two pleural cavities and, to a great extent, on the anatomic status of the mediastinal and pleuromediastinal connective tissue. This varies constitutionally, by scar formation it may become firm or it may loosen and become weak from acute inflammation.

Parenthetic reference should be made to the herniation of the anterior and posterior weak area of the mediastinum (Brauer) by a pneumothorax, this is not uncommon. Apart from other implications which need not be discussed in this place such mediastinal herniations explain some cardiac complaints such as pain and disturbances of rhythm (fig. 162)

With pleural effusions, and particularly with pneumothorax, in inspiration the mediastinum wanders toward the side of the effusion or pneumothorax, naturally, alterations of cardiac shape result.

Adhesions of the mediastinal pleura often distort the entire mediastinum, fill the cardiodiaphragmatic angle, and cause vague, irregular delineation or local traction on the cardiovascular borders By extension to the mediastinal connective tissue and the pericardium, they may fix the heart to the anterior chest wall, the spine, or lungs so that the clinical manifestations of accretio cordis appear (p 332). With deep inspiration, the heart frequently rises with the anterior chest wall or moves to the side of the adhesions.

Very frequently with indurative processes in the lungs (above all with tuberculosis and pneumoconiosis) which are ordinarily associated with considerable emphysema, both borders of the mediastinal shadow are stretched so that the cardiovascular shadow rests on the diaphragm like a small, median, equilateral triangle A similar distortion is produced by contraction of a pleuromediastinal adhesion

In inspiration pleuromediastinal scars may pull the entire heart to the affected side and stretch the cardiac border.

Distortion of the heart and great vessels may be very striking with shrinking processes in the lungs Extreme retraction may draw the heart to the lateral or posterior chest wall (Wiese) so that the entire spine is exposed and the heart may vanish completely within the pulmonary shadow Then, exact delineation of the heart is absolutely impossible (fig. 163), it is astonishing how such distortions of the heart and of large efferent and afferent vessels, create no circulatory disturbances and how well function is retained Especially marked cardiac distortion is found with skrinkage of the lungs acquired early in life, particularly with indurative processes accompanying bronchiectasis

Shrinking processes in the left upper lobe or left hilus often elevate the pulmonary artery so that the cardiac shadow acquires a mitral configuration (fig 159).



FIG. 163.—Marked distortion of the mediastinum through contraction of the left bronchiectatic lung. The trachea and bifurcation (white arrows) are pulled far into the left half of the thorax. The feathered black arrows point to the boundary of the right lung drawn into the left hemithorax. The dark arrow directed to the right indicates the left border of the descending aorta No circulatory disturbances.



a



b

FIG. 164—Marked retraction of the heart to the right and posteriorly by bronchiectatic shrinkage of the right lung Female, 49 years old. (*a*) Anterior view. The arrows indicate the course of the trachea. (*b*) Right lateral view. The heart is drawn away from the anterior chest wall and is broadly superimposed by the left lung

This distortion of the pulmonary artery may dissociate the valves and cause incompetence (p. 240).

Marked retraction of a lower lobe occasionally pulls the entire mediastinum backward so that the heart is situated alongside the spine and is widely superimposed by the other lung, distended from vicarious emphysema (Giordano). In lateral views, the heart is largely or completely projected into the vertebral column (fig. 164a and b).

Indurations of the right upper lobe and of the right hilus as well as right-sided mediastinal pleural scars often distort the ascending aorta. Then, the aorta projects farther to the right, often as a rounded or angular shadow. In this way the picture may simulate somewhat a dilated ascending aorta (p. 386). Measurement of the ascending aorta, and particularly the demonstration of right-sided pleuromediastinal adhesions or a shrinking process in the right upper lobe, as a rule permits the differentiation (fig 165).

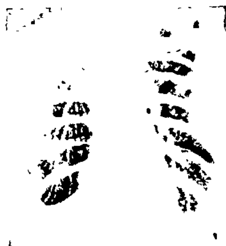


FIG. 165.—Traction on the ascending aorta by a shrinking process in the right upper lobe.

X. The "Gastrocardiac Complex"

In this conception (Romheld) are included certain cardiac complaints like pain, feeling of compression and disturbances of rhythm which are not produced by cardiovascular disease but indirectly or directly by the gastrointestinal tract. Mechanical as well as reflex influences on the heart are responsible but intestinal intoxication has also received etiologic consideration

The gastrocardiac symptom complex is found chiefly in pyknic males in the fifth and sixth decades Their build, well developed obesity and good tonus of the abdominal wall, elevates the diaphragm and displaces the heart transversely The complaints are released by gastric distension or by constipation and meteorism but often no precipitating causes are discovered.

The distress is sufficiently annoying to make the patient despair and it may have an anginal component Corresponding to the transverse position and elevation of the heart and great vessels, cardiac dullness is broad. Frequently the blood pressure is moderately elevated. If a systolic murmur is heard over the aorta or the second aortic sound is accentuated, it is easy to assume that an atheromatous heart or syphilitic aortitis with coronary artery damage is present.

Examination reveals a transverse elevated heart of normal size although the marked rounding of the left ventricular arc may indicate left ventricular hypertrophy

in hypertensive patients. Since the aortic loop is displaced upward, the vascular band is broad (p. 357), creating an impression of aortic dilatation in the anterior view. Only measurement of its diameter shows that the aorta is normal or slightly wide in keeping with the advanced age or the moderate hypertension present in many of these patients.

Occasionally the large bowel is markedly distended At times there is a unilateral but more often bilateral elevation of the diaphragm (fig. 166) produced by distention of the splenic flexure or a large gastric gas bubble (aerophagia). At times a left diaphragmatic hernia or paresis explains the complaints. Von Bergmann has

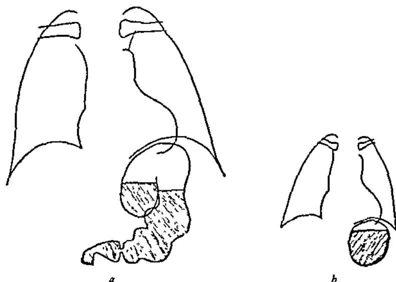


FIG 166.—Gastrocardiac syndrome with left sided elevation of the diaphragm and cascade stomach. Male, 36 years old with normal electrocardiogram On standing, at first only the cephalic portion of the stomach (*b*) fills In this position a marked sense of oppression. Only in the right lateral position does the gastric content appear in the caudal part of the stomach (*a*). Then the patient felt relieved

referred to hiatus incompetence when luxation of the cardiac part of the stomach above the level of the diaphragm produces angina (fig 167).

The causal connection between the complaints and the various situations demonstrated roentgenologically remains uncertain in many cases, examination, significant only in so far as it reveals some conditions, does not disclose why cardiac oppression, pain, extrasystoles, and tachycardia are released when the coronary circulation is completely normal Perhaps these conditions favor the appearance of true angina pectoris in patients with abnormal coronary circulation. A left or bilateral elevation of the diaphragm, a diaphragmatic hernia, or diaphragmatic or hiatal incompetence in no way precludes the existence of angina pectoris produced by organic coronary artery disease. In this connection it may be recalled that elevation of the left diaphragm, abnormal distention of the stomach or bowel by gas, or even a left diaphragmatic weakness may result from myocardial infarction (Borak, Laubry) (p. 229).

Electrocardiograms have great value in determining whether the angina pectoris of these patients depends upon coronary artery disease or not, although a normal tracing does not absolutely exclude disease of these vessels (Scherf, Jagič and v. Zimmermann-Meinzingen). *Whether or not the situations mentioned above are adjuvant factors in the appearance of myocardial hypoxia or whether they are actual results of coronary artery damage often cannot be decided despite a most careful history and recourse to all clinical measures.*

Whether hiatal insufficiency has causal significance in defective myocardial perfusion (v. Bergmann) is dubious. Although animal experiments provided some evidence favoring this conception (Dietrich and Schwiegk), the studies of Zdansky

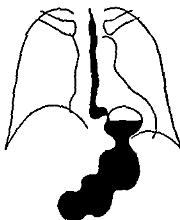


FIG 167 —Gastrocardiac syndrome with hiatus hernia. Male, 59 years old For two years he had suffered from complaints like angina pectoris, especially after large meals Nitroglycerin did not relieve the pain Electrocardiogram and blood pressure normal Wassermann negative.

and Ellinger supplied no support for the assumption of a relation between hiatal weakness and angina pectoris in the sense of an "epiphrenic syndrome"

XI. The Heart in Thoracic Deformities

With severe kyphoscoliosis of the thoracic spine, cor pulmonale is not unusual. Consequently, clinical signs of severe circulatory failure often appear and may prove fatal in a relatively short time, the syndrome consists of extreme dyspnea, cyanosis, and retrograde stasis in the systemic circulation.

The demarcation of the cardiac shadow on all sides is very difficult since the heart is, for the most part, projected into the dense shadows of the curved spine or deep into the abdominal shadow. The latter happens particularly when the diaphragm descends from back to front so that the heart sinks far into the angle formed by the diaphragm and the anterior chest wall

Despite these difficulties examination usually reveals that the caval axis of the *heart does not tend to follow the lateral deviation of the spine with high grade* scoliosis but remains as nearly perpendicular as possible (fig. 168) There-

fore, the position of the entire heart in the thorax does not shift as much as one might expect from the extent of chest deformation. Nevertheless, shortening of the thorax often makes the heart transverse and causes greater bulging of the left cardiac border and deepening of the cardiac waist, consequently a majority of patients with kyphoscoliosis have an aortic configuration (Groedel, Amelung, Dietlen). Sometimes this is accentuated by left ventricular hypertrophy when the aorta is kinked markedly owing to the chest deformity (fig 168).

In pure kyphoscoliosis, the effects of a shorter and deeper thorax are strongly reflected in the position of the heart. In a posteroanterior view, the heart sinks deep into the abdominal shadow so that it appears smaller than it actually is. Its true size is appreciated only in lateral views; then, one sees how the heart is broadly



FIG. 168.—Kyphoscoliosis of thoracic spine.

applied to the anterior chest wall and to the diaphragm which is more or less flattened and descends from behind and above to front and below (fig. 169a and b). The inclination of the heart from behind and above to below and in front may be 20 degrees or less.

The deep cyanosis, severe dyspnea and peripheral stasis of individuals with kyphoscoliosis often stands in striking contrast to the slight enlargement or even normal size of the cardiac shadow. This contrast is understandable when one recalls that circulatory failure—just as in many patients with emphysema—often does not depend upon cardiac weakness but on incompetence of the diaphragmatic and costal forces and reduced amplitude of respiration (Wenckebach, Hofbauer). The minimal respiratory excursions of the perfectly flat diaphragm, visible on the screen, provide an impressive picture of the profound involvement of respiration.

In all kyphoses and kyphoscolioses the aortic arch moves toward the spine. Often considerable elongation and an entirely abnormal course of the aorta results;

for example, the vessel may be directed posteriorly and to the right. Angular aortic kinking is not uncommon (fig. 169b).

The close spatial relationship between the arch of the aorta and the esophagus remains unchanged in most cases, almost always Kreuzfuchs' measurement can be performed. Nevertheless, there are cases in which the aortic bed is lower than the most lateral point of the aortic knob, this prevails with high grade kyphosis when the aortic arch ascends upward and backward to the spine beyond the site of crossing of the esophagus (H. Rosler) In general, the course of the spine affects the esophagus less than the aorta, the esophagus never completely follows spinal curvature but either bends moderately in the direction of the scoliosis or descends caudad in a perfectly straight line.

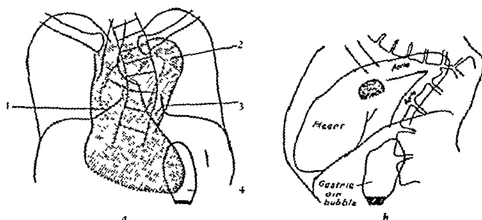


FIG 169—Thoracic deformity by gibbus in midthoracic spine Relatively little alteration of cardiac position On the contrary, the aorta which has moved toward the spine shows an angular kink at the level of the gibbus (*a*) Anterior view, (*b*) left lateral view

1 Lumbar part of diaphragm
2 Aorta
3 Lumbar part of diaphragm
4 Gastric bubble

Pulmonary congestion and edema are not a part of the picture of kyphoscoliosis, although *they appear in terminal stages. Accordingly, the lung fields are generally clear unless chest deformity renders them hazy*

Slight deformation of the chest exerts no injurious influence on the function of the heart and circulation but changes the position and shape of the heart as will now be discussed

The common, slight spinal curvature convexly to the right displaces the entire cardiovascular complex *to the left so that the left border of the cardiac shadow may* extend almost to the lateral chest wall As the pulmonary arc projects farther, the cardiac waist becomes shallow and a mitral configuration *becomes more or less* obvious (fig 170a). Brugsch observed bulging of the pulmonary arc in 80 per cent of all scolioses and considered it a stigma of a hypoplastic degenerative cardiac anlage, Amelung noted this bulging in only 30 per cent of his cases. The incidence reported by Brugsch as well as his interpretation has *received much criticism (Dietlen,* Assmann, Rosler), in particular Assmann pointed out that the mitral configuration may result from right ventricular hypertrophy. On *the contrary* we believe that

trifling spinal curvatures can hardly induce such hypertrophy and concur with Rosler that bulging of the pulmonary arc in these cases results from cardiac rotation to the left consequent to altered intrathoracic spatial conditions. *It is easy to make* the mitral configuration vanish by turning the patient to the left far enough to offset the cardiac rotation and until the spine runs near or in the middle of the cardiovascular shadow (fig. 170b).

Scoliosis of the thoracic spine, convex to the left, is much less common than to the right. With it, most of the cardiovascular complex lies to the right of the spine so that the left cardiac border protrudes less and the cardiac shadow seems rather *median*. With scoliosis of the upper and middle third of the thoracic spine, the aortic knob projects less but the entire vascular band is more or less widened since the

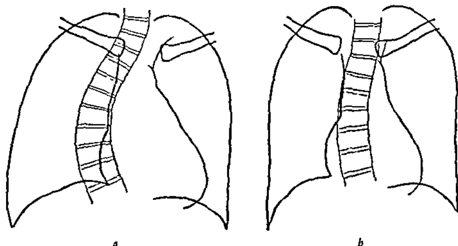


Fig. 170.—Mitral configuration of the heart with convex scoliosis of midthoracic spine to the right (*a*) Anterior view, (*b*) by slight rotation of patient to right normal configuration is restored (copies of films).

descending aorta follows the spine and is exposed on the left (H. Rosler). When the scoliosis to the left is sufficiently marked, occasionally the whole loop of the aorta is visible in the posteroanterior projection, a picture normally seen only in the left anterior oblique position

Naturally the close spatial connection between the aortic arch and the esophagus is ordinarily preserved with slight spinal curvatures so that Kreuzfuchs' aortic measurement is usually free from technical objections.

With deep respiration mediastinal wandering is occasionally noted, in inspiration it is directed toward the side of the concavity of the spinal curvature (Zdansky).

is never observed.

Funnel chest influences cardiac shape and size by reducing the sternovertebral diameter. This can amount to 3 cm or less, in one case it was only 1.3 cm (Rosler). Nevertheless, cardiac function is scarcely embarrassed. As a rule the heart escapes compression by more or less evasion to the left (Groedel), in this way the right border can be projected into the spinal column while the left can extend almost to the left chest wall (fig. 171a and b). With less severe funnel chest, the heart is merely flattened in a dorsoventral direction Then, with posteroanterior projection the cardiac shadow appears more plump and enlarged to left and right Frequently the conus pulmonalis projects into the cardiac waist so that a mitral configuration appears (fig. 172). Since these patients frequently have a systolic murmur over the heart, the inexperienced student may be misled into thinking a mitral lesion is



Fig 171 —Deformation of heart in funnel chest (*a*) Anterior view. The heart is displaced to the left and is radiolucent in its center from compression in ventrodorsal direction (*b*) Lateral view The heart, displaced into the left half of the chest, projects ventrad behind the posterior aspect of the sternum (arrow)

present The demonstration of a normal left atrium should, however, prevent the mistake Corresponding to the cardiac indentation through the trough-like depression of the sternum, a central clearing can sometimes be seen in the anterior view (Pohl) (fig 171). With lateral projections the heart often projects distinctly in front of the posterior surface of the sternum

Severe funnel chest is often associated with spinal scoliosis, concave to the right, whereby the heart again gains more space. This scoliosis can also influence the shape of the cardiac shadow in various ways

In funnel chest the esophagus may follow its normal course, deviate to the left or, more rarely, to the right.

Pure lordoses of the middle and lower spine are rather rare and usually result from a gibbus of the upper section of the column. These lordoses can lead to a funnel-like depression on the back which also reduces the sternovertebral diameter of the

trifling spinal curvatures can hardly induce such hypertrophy and concur with Rosler that bulging of the pulmonary arc in these cases results from cardiac rotation to the left consequent to altered intrathoracic spatial conditions. It is easy to make the mitral configuration vanish by turning the patient to the left far enough to offset the cardiac rotation and until the spine runs near or in the middle of the cardiovascular shadow (fig. 170b).

Scoliosis of the thoracic spine, convex to the left, is much less common than to the right. With it, most of the cardiovascular complex lies to the right of the spine so that the left cardiac border protrudes less and the cardiac shadow seems rather median. With scoliosis of the upper and middle third of the thoracic spine, the aortic knob projects less but the entire vascular band is more or less widened since the

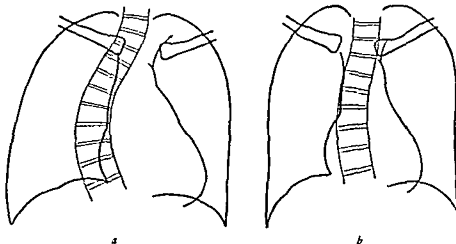


Fig. 170.—Mitral configuration of the heart with convex scoliosis of midthoracic spine to the right. (*a*) Anterior view, (*b*) by slight rotation of patient to right normal configuration is restored (copies of films).

descending aorta follows the spine and is exposed on the left (H Rosler). When the scoliosis to the left is sufficiently marked, occasionally the whole loop of the aorta is visible in the posteroanterior projection, a picture normally seen only in the left anterior oblique position.

Naturally the close spatial connection between the aortic arch and the esophagus is ordinarily preserved with slight spinal curvatures so that Kreuzfuchs' aortic measurement is usually free from technical objections.

With deep respiration mediastinal wandering is occasionally noted; in inspiration it is directed toward the side of the concavity of the spinal curvature (Zdansky). Awareness of this wandering is important to avoid the erroneous assumption of a bronchial stenosis. The ribs on the side of the concavity have a greater radius than those on the side of the convexity; accordingly with inspiratory costal elevation, the side of the concavity experiences a greater increase of capacity and a greater reduction of intrathoracic pressure than the opposite side. This may draw the mediastinum to the side of the concavity. With severe kyphoscoliosis, this mediastinal wandering is never observed.

cardiac stab and gunshot wounds, *death soon occurs owing to tamponade or exsanguination.* Nevertheless, it is not uncommon for a stab or gunshot wound in the ventricular wall to be so narrow that blood enters the pericardial cavity slowly and cardiorraphy undertaken at the proper moment may be lifesaving. In these cases physical examination often fails to reveal unequivocally whether or not the heart is injured. By portable apparatus at the bedside or in the operating room, however, the positive demonstration of a pericardial effusion immediately establishes the diagnosis of a cardiac or pericardial injury. Often in these injuries a pneumo- or hydropneumothorax is found, since concomitant pleural injury is common. Occasionally air entering from the outside or from the pleura into the pericardial cavity causes a hydropneumopericardium (p. 329).

Lacerations of the heart valves *from chest wall trauma (crushing, gunshot,* explosions) are rare. When simultaneous injuries of the myocardium or other organs do not prove fatal, such tears may lead to permanent incompetence of the valves or to stenosis from scar formation so that the clinical and roentgenologic picture of a valve lesion develops, this need not differ from one of rheumatic or syphilitic origin (Kienbock, Gussenbauer, Luthi).

Somewhat more common are hemorrhages or necrotic foci in the myocardium from indirect force ("contusio cordis" of Hadorn and Tillmann), the genesis is not entirely clear and probably is not uniform. Such foci can lead to cardiac dilatation from myocardial failure, to the formation of a cardiac aneurysm or to belated rupture and tamponade (Dietrich).

Experimental studies on animals indicate that blunt injuries of the chest wall which do not directly involve the heart can precipitate acute cardiac dilatation even without demonstrable myocardial damage (Schlomka and coworkers) These dilatations may disappear in minutes or hours or they may be followed by fatal "secondary dilatation" The extent of dilatation experimentally produced roughly parallels the severity of the blunt force This "commotio cordis" is accompanied by profound electrocardiographic changes as well as by a fall of the arterial and a rise of venous pressure, frequently there is an associated bradycardia, tachycardia, extrasystoles, ventricular flutter, or fibrillation. In rabbits previously treated by a foreign serum there may be circumscribed anemic zones in the ventricular muscle while the atria are engorged. Schlomka ascribes these alterations to a disturbance of myocardial *perfusion resulting from coronary spasm*

Similar posttraumatic, even very transient alterations, seem to occur in man. Some clinical observations suggest it Apparently no roentgenologic observations are available at present

Projectiles, splinters of bullets, or other metallic foreign bodies can penetrate the pericardium, heart wall, or cardiac cavity Their roentgenologic demonstration has great practical importance

If the missile has merely penetrated the pericardium, it may be associated with a hemopericardium from injury of a pericardial or myocardial blood vessel or, with the entrance of air from a coincident pneumothorax or of outside air, with a pneumopericardium. One sequela of such hemorrhage is pericardial adhesion or mediastinopericarditis (p 332) (Gaisbock) Pericardial injuries without hemorrhages are also known.

chest. Although complaints are infrequent (Edeiken), the heart can be flattened by this compression (fig. 173).

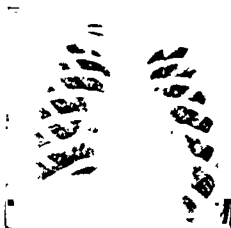


FIG 172.—Deformation of the heart in funnel chest. Ventrodorsal compression of the heart has erased the cardiac waist and has resulted in a mitral configuration.

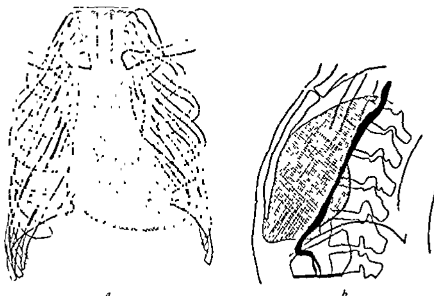


FIG. 173 —Ventrodorsal compression of heart by lordotic curvature of midthoracic spine (copy of film). Old male with gibbus in uppermost thoracic spine Owing to reduction of the sterno-vertebral diameter, the heart is spread and projects—as the lateral view (*b*) shows—over the spine dorsad on the left side No cardiac complaints

XII. The Heart and Trauma

Rupture of the heart from crushing or bursting is not subject to roentgenologic investigation. Usually this results from severe chest injuries. Moreover, in most,

Trendelenburg, Ledoux-Lebard, Becher, and others observed whirling and dancing movements of intracardiac projectiles. Rosler also depicted circular movements in which the bullet slid along the ventricular septum toward the base of the heart in systole and more slowly returned along the outer wall to the apex in diastole

If a freely movable projectile within the heart is not ejected into one of the vessels, usually it soon becomes fixed in the atrium or between the trabecula of the ventricle (Ascoli, Becher, Rosler) Fixation in the wall of a chamber may be permanent, but the object may loosen later and escape as an embolus (Kienbock)

Paracardial fragments in the anterior or posterior mediastinum or alongside the heart can cause numerous complaints like pain, oppression, tendency to collapse, and disturbances of rhythm through scars involving the pericardium or heart or by injury to the networks of vegetative nerves. X-ray allows one to determine the precise relation of the opaque foreign body to the heart.

XIII. Calcification of the Valves and the Cardiac Skeleton

Intracardiac calcification is very common, usually it involves the valves, the cardiac skeleton, or both Calcification of the coronary arteries, of the myocardium, or of large parietal thrombi are numerically in the background when compared to those mentioned first; they have been discussed on page 225.

Most valvular calcifications develop on the basis of a chronic rheumatic endocarditis and more rarely from primary degenerative changes in the tissues composing the valve

In the first instance calcification occurs primarily at the edge of the valve but it may advance down to its attachment and even into the endo- and myocardium. Any ostium may be involved but, corresponding to the frequency of mitral and aortic valvulitis, preference is given to these sites

In the second group, calcification is usually associated with similar alterations in the cardiac skeleton from which they tend to arise. By cardiac skeleton one understands the connective tissue portion of the heart inserted between the atrial and ventricular musculature It serves for the attachment of the atrioventricular valves and as sites of origin for the large vessels, it consists of poorly nucleated connective tissue and surrounds both atrioventricular ostia with the right and left annulus fibrosus. The annuli unite to form a large connective tissue plate which widens into the right and left trigonum fibrosum The bundle of His passes through the right trigonum To the left of the bridge connecting the two trigona is the anterior (aortic) leaflet of the mitral ostium, to its right is the posterior and the left semilunar valve of the aortic ostium.

Calcifications of the cardiac skeleton develop on the basis of fatty degenerative or necrotic processes and are usually found first at advanced ages They are rare in the relatively well-nucleated trigona, of these the left annulus fibrosus and the valvular ring of the aortic ostium are affected most often, involvement of the right annulus and of the ring of the pulmonary valve is relatively rare

In the left annulus fibrosus, calcification usually begins posteriorly to advance to the valves or toward the apex in the ventricular septum. Thus, a ring, the thick-

According to the position of the patient and of a metallic foreign body, the object can be projected on the cardiac surface or into its depth. For precise localization, fluoroscopy in many positions is indispensable. A projectile lying in the pericardial cavity must be projected on the cardiac surface in at least one position. If freely movable in the cavity its location may change with a new position of the body (Kienbock) although fixation by intrapericardial coagulum or adhesions is customary. If embedded in a cicatrix it may be impossible to decide whether it is intrapericardial, just outside the pericardium or actually on the cardiac surface. Corresponding to its relation to the cardiac surface, it participates in the pulsations of the adjacent section of the heart.

Many descriptions of missiles implanted in the heart are available. The subject of localization by careful fluoroscopy in various planes has been reviewed by Kienbock and by Steffens. They, as well as Freund and Caspersohn, Kukula, Becher, Franchini, v Zezschwitz, and others, describe the striking movements of such objects. Corresponding to the favored position of implanted bullets in the ventricular musculature, the pulsations are usually ventricular, that is, they are synchronous with systole even if not always identical with visible ventricular pulsations. According to the position and plane of projection, a foreign body usually shows parallel shifts or circular movements with remarkable variations in the amount of excursion. It may be 1 to 2 mm. or 2 cm. and more Von Zezschwitz observed a fragment of shot fluoroscopically, it was located near the atrioventricular septum and shifted 3 cm. systolically toward the apex, this illustrates excellently the extent of systolic shift of the atrioventricular septum (Laurell). Occasionally the missile rotates around a point of fixation (Freund and Caspersohn). In this instance the object is fixed in the pericardium and projects partly into the heart muscle; such rotation can also occur when the parts of the heart through which the shot passed contract to different degrees and in different directions.

Projectiles in the anterior cardiac wall usually shift to the right in systole since the heart rotates to the right at this time.

In addition to movements synchronous with the heart, the projectile may also shift with respiratory changes of cardiac position.

Bullets and fragments of shot inside the cardiac cavities can arrive directly through the myocardium or from the great veins.

Direct penetration may lacerate the valves and create corresponding clinical and roentgenologic signs (Kienbock, Gussenbauer, Luthi). Bullets entering from the veins usually start from an abdominal wound, especially from the liver, to pass through the inferior vena cava and reach the right heart (Freund and Caspersohn, Ascoli, McCartney and Drummond, Debreye and Lorgnier, Duval and Barnsby, among others). We found a fragment near the tricuspid ostium which had entered through the left innominate vein.

The projectile may escape from the heart into the pulmonary artery (Kukula, Bishop) or through the aorta into the carotid (Luthi), the subclavian (Kienbock), the femoral (Boeckel) or other peripheral arteries. Moreover, retrograde passage from the right heart into the hypogastric or femoral veins is known (Rosler). Owing to the possibility of migration it is essential to be sure that the object is still in the heart just before an attempt at operative removal is made.

Calcifications of the cardiac skeleton can produce very similar shadows when they extend to the valves (fig. 177). If they are limited to the cardiac skeleton, however, the shadows are thick, sharply defined, and ring or sickle shaped (figs. 178a and b and 179a and b) (Klason, Fleischner, Saul, Parade and Kuhlmann, Baumann and Naumann, Bishop and Rosler, Kommerell).

With dorsoventral projection, mitral calcifications are projected into the middle and basal thirds of the cardiac shadow, 0.5 to 2 0 cm. to the left of the spine, the aortic valve is somewhat higher and usually near enough to the median plane to lie in the dark shadow of the vertebral column To demonstrate them the patient should be rotated into the left anterior oblique position to throw the calcification to the left and out of the spinal shadow.

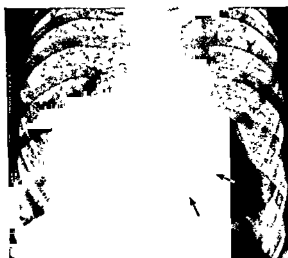


FIG 175 —Calcification of mitral leaflet in mitral stenosis Female, 26 years old The visible calcifications (arrows) executed systolic excursions toward the base of the heart

With dextrosinistral projection, naturally calcifications are less demonstrable because of slight opacity; however, in this position it is much easier to determine whether they belong to the mitral or aortic ostium since the mitral lies lower in the middle or dorsal third of the cardiac shadow while the aortic is higher in the ventral third.

Pulsa

shadow. .

of projec

triangular and have a jerky, dancing character since the velocity of the rotatory movement is not uniform In general, movement toward the apex is more brisk during systole and slower toward the base in diastole, that is, the excursions follow movements of the atrioventricular septum. By roentgenkymography Odquist demonstrated a brief vibratory phase just before attaining the vertex of the upward movement in annulus calcification, he interpreted this as upward retraction of the

ness of a finger, can surround the mitral ostium. By retraction within the involved valves, the ostium may become stenotic and the valve incompetent.

In the aortic valves, calcification usually begins at the base to advance to the commissure, the outer side of the valves and the aortic root so that a nodular excrescence gradually fills the sinus of Valsalva. By shrinkage the aortic ostium may become stenotic. Usually the coronary ostia are not affected.

Calcification of the mitral valve is most common, limitation to the aortic ostium is rarest for these are less frequent than combined calcifications of the mitral and aortic ostia (Giese).

When the right trigonum fibrosum is affected, conduction disturbances such as partial or complete heart block and prolonged conduction time may result.



Fig. 174.—Calcification of a mitral leaflet in mitral stenosis (necropsy). Male, 43 years old. The calcium deposit, visible in the region of the mitral ostium (arrow) executed systolic pulsations toward the base of the heart, this revealed its location in the mitral leaflets.

Naturally calcifications of the valves or cardiac skeleton are demonstrable only when the deposits are large, the heart is not too big and the patient not too obese. They are easiest to discover when fluoroscopy is performed with hard light, and a narrow diaphragm to prevent secondary radiations. Pulsations facilitate visibility on the screen. Sharp pictures can be expected only on shielded films taken with very short exposures.

Valvular calcifications after rheumatic valvular lesions are more common than one might expect from roentgenologic literature. However, Parade and Kuhlmann, Sosman and Wosicka, Sparks and Evans, and Baumann and Naumann, as well as Kommerell and many others, described cases. We have seen and confirmed at necropsy many examples. Usually, demonstrable valvular calcifications follow mitral endocarditis (figs. 174 and 175) and more rarely aortic valvulitis (fig. 176). They are seen as lumps or spotted shadows, arranged singly or in irregular crescents or as ring-shaped groups (figs. 174 to 176).

atrioventricular septum by the presystolic atrial contraction. More rarely the calcareous mass moves toward the base in systole. Probably this movement indicates that the calcification involves the valve leaflet since the valve is thrust basal in systolic closure; exceptions exist as necropsy has proved. The movements of the mitral ostium during cardiac activity are extremely complicated. They include systolic wandering of the atrioventricular septum toward the apex and tilting of the valve ring rhythmic with cardiac contraction, this varies greatly with the position of the heart in the chest. Roentgenologically, this tilting may move the calcified annulus opposite to the systolic septal wandering. In roentgen views, once again, calcifications of the valve leaflets frequently show a systolic excursion toward the apex,



Fig. 179 —Calcification of the annulus fibrosus sinistra. Female, 79 years old, without signs of valvular lesion The buckle-shaped calcium deposit (arrow) moved toward the apex in systole. (*a*) Anterior view, (*b*) left anterior oblique view.

since the valve is fastened to shortened tendons and tends to be incapable of closure, it must participate in movements of its ring.

Through their location and pulsations, calcifications of the valves and cardiac skeleton are differentiated from other intracardiac calcifications Among these are thrombi, originally attached to the cardiac wall like a mushroom, and "cardiac polyps," which occasionally calcify and become demonstrable by x-ray They are located in the ventricles between the trabeculae especially near the apex, in the auricular appendages, or in valve remnants in the atrial septum, in the latter instance they are found at the *Valv. foraminis ovalis* on the left side and on the right at the *Valv sinus coronarii* (Thebesi). Besser and Schilling, Heeren, and Berk and Rauch reported the roentgen findings of calcified, spheric thrombi in the left and right atria. The nodular or almost spheric clumps, ring or half-moon shadows, corresponding to their position, can be projected just to the cardiac surface In contrast to the pulsations mentioned above, these excursions never extend beyond the cardiac surface. In a 72 year old woman with essential hypertension, Zdansky saw a cherry-sized, cup-shaped shadow in the hypertrophied and enlarged left ventricle (fig. 180a



Fig. 176



Fig. 177

Fig. 176.—Extensive arteriosclerotic calcification of aortic valves and of aorta. A crescent shaped mass of calcium is seen in the sinus of Valsalva (arrow).

Fig. 177.—Ring-shaped calcification of annulus fibrosis and the leaflet of the mitral ostium in a combined mitral-aortic lesion. Male, 49 years old. Systolically the flakes of calcium moved toward the apex



a



b

Fig. 178.—Calcification of the annulus fibrosus sinistra Female, 70 years old, without signs of valvular lesion. The calcific, semicircular shadow (arrow) moved toward the apex in systole (a) Anterior view, (b) right anterior oblique view

until recently, rather meager. Moreover, the roentgen symptomatology was somewhat rudimentary despite the publication of several works (Assmann, Vaquez and Bordet, H. Muller Sr. and Jr., Blumenfeldt, P. White, Roesler, Zdansky, and others). In recent years, however, significant advances have been made in the diagnosis of these anomalies These advances stem primarily from Abbott's thorough pathologic studies, the fundamental clinical work of Taussig, and the development of angiocardiography by Castellanos and coworkers as well as by Robb and Steinberg. Interest in the diagnostic aspect also received a powerful stimulus by virtue of the progress in surgical management (Blalock and Taussig, Gross, Crafoord and Nylin, Brock). By the assembly of a large number of appropriate cases in centers which subsequently became widely known for cardiac surgery, it was possible to supplement the fragmentary knowledge of clinical and roentgenologic diagnosis by experiences derived from plentiful material

At the outset it may be stressed by way of anticipation that in no section of diagnostic cardiology do roentgen findings alone provide reliable conclusions on the nature of existing alterations, this situation also prevails in respect to congenital malformations. Even those anomalies which profoundly hamper activity and which are accompanied by deep cyanosis can yield normal or completely indeterminate roentgen findings. On the other hand, there are cardiovascular anomalies whose abnormal structure is immediately apparent or whose perverse dynamics functionally create anatomic alterations which completely dominate the roentgen picture. Although the functional reactions of the heart fundamentally follow the same laws as those prevailing in the heart of normal structure, still they exhibit many special features; these can be explained partly by the working conditions in fetal or, more commonly, in neonatal life, differing from those existing in acquired disorders; they are also explained in part by the architecture of single cardiac sections which deviate from normal. Consequently, deformations of the cardiac shadow can occur which exhibit such striking differences from those seen in acquired lesions that the probable diagnosis of a congenital anomaly is reached immediately; in other cases, the forms may simulate acquired cardiac pathology so closely that unawareness of the congenital nature and the clinical manifestations make a correct interpretation of the roentgen findings impossible. Through this interweaving of deviations of cardiac shape dependent upon abnormalities of the developmental plan and the anatomic consequences of abnormal function and altered intracardiac dynamics, malformations due to perverse and arrested development, differing greatly from an anatomic and evolutionary standpoint, may have great similarity while closely related anomalies which form a series by small gradations may show extreme differences. Consequently the roentgen pictures of congenital malformations should be interpreted only in conjunction with the total clinical picture and with knowledge of the case history, moreover sweeping conclusions should be avoided.

The clinical and roentgenologic diagnosis is rendered more difficult by the fact that inflammatory processes in the valvular apparatus frequently complicate the situation The value of a careful clinical examination including the electrocardiogram in conjunction with the roentgen findings is evident from the excellent work of Taussig and from recent statements by Méteanu, Dubost, Durand and Hoffmann. Moreover, angiocardiography is indispensible in a great many cases to provide

and b), it showed systolic pulsations directed dorsocraniad in the region of the atrial septum. Corresponding to this position and pulsation one must assume that a calcified parietal thrombus or varix was present. Occasionally varices form in the region of the fossa ovalis (Frank, Nauwerck); if filled with thrombi, they may calcify as phleboliths or more extensively. To be sure, no case thus diagnosed has been confirmed at necropsy. Ball thrombi also are still not described in roentgenologic literature. Calcified echinococcus cysts can be distinguished from thrombi only

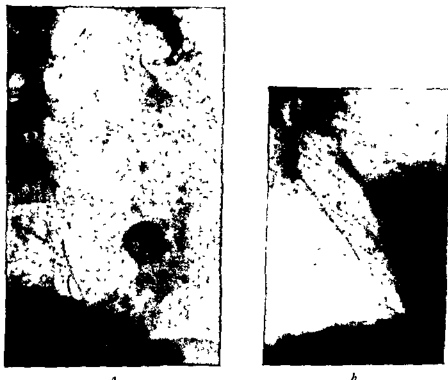


FIG 180—Calcified parietal thrombus or varix in atrial septum. Female, 72 years old, with essential hypertension. In the region of the atrial septum of the heart which was hypertrophied and dilated on the left side, there is an oval shadow which showed systolic pulsations directed dorso-cephalad (*a*) Right anterior oblique view, (*b*) right lateral view.

when the cysts protrude beyond the cardiac surface; then, of course, their location in the ventricular wall makes one think of calcification in a cardiac aneurysm.

Calcification of coronary arteries (p. 226) is rarely demonstrable, it could hardly be confused with calcification of the valves or the cardiac skeleton. Calcification of pericardial adhesions (p. 345) also is not a likely source of error.

XIV. Congenital Malformations of the Heart

Since these anomalies represent about 0.7 per cent of all cardiac lesions (Gerhardt) they have a definite role in cardiac pathology. Most of them are incompatible with long life so that relatively few of those affected reach childhood or adult life. Consequently, the experiences assembled by individual clinicians in this field were,

until recently, rather meager. Moreover, the roentgen symptomatology was somewhat rudimentary despite the publication of several works (Assmann, Vaquez and Bordet, H Muller Sr. and Jr., Blumenfeldt, P. White, Roesler, Zdansky, and others). In recent years, however, significant advances have been made in the diagnosis of these anomalies These advances stem primarily from Abbott's thorough pathologic studies, the fundamental clinical work of Taussig, and the development of angiocardiography by Castellanos and coworkers as well as by Robb and Steinberg. Interest in the diagnostic aspect also received a powerful stimulus by virtue of the progress in surgical management (Blalock and Taussig, Gross, Crafoord and Nylin, Brock). By the assembly of a large number of appropriate cases in centers which subsequently became widely known for cardiac surgery, it was possible to supplement the fragmentary knowledge of clinical and roentgenologic diagnosis by experiences derived from plentiful material.

At the outset it may be stressed by way of anticipation that in no section of diagnostic cardiology do roentgen findings alone provide reliable conclusions on the nature of existing alterations, this situation also prevails in respect to congenital malformations Even those anomalies which profoundly hamper activity and which are accompanied by deep cyanosis can yield normal or completely indeterminate roentgen findings. On the other hand, there are cardiovascular anomalies whose abnormal structure is immediately apparent or whose perverse dynamics functionally create anatomic alterations which completely dominate the roentgen picture. Although the functional reactions of the heart fundamentally follow the same laws as those prevailing in the heart of normal structure, still they exhibit many special features; these can be explained partly by the working conditions in fetal or, more commonly, in neonatal life, differing from those existing in acquired disorders, they are also explained in part by the architecture of single cardiac sections which deviate from normal. Consequently, deformations of the cardiac shadow can occur which exhibit such striking differences from those seen in acquired lesions that the probable diagnosis of a congenital anomaly is reached immediately, in other cases, the forms may simulate acquired cardiac pathology so closely that unawareness of the congenital nature and the clinical manifestations make a correct interpretation of the roentgen findings impossible Through this interweaving of deviations of cardiac shape [illegible] abnormalities of the developmental plan and the anatomic conse[illegible]
due t[illegible]
evolu[illegible]
which form a series by small gradations may show extreme differences Consequently the roentgen pictures of congenital malformations should be interpreted only in conjunction with the total clinical picture and with knowledge of the case history, moreover sweeping conclusions should be avoided.

The clinical and roentgenologic diagnosis is rendered more difficult by the fact [illegible]

[illegible]
Moreover, angiocardiography is indispensible in a great many cases to provide

better insight into the size, position, and shape of single divisions of the heart and information about abnormal communications which may be present. To be sure, not uncommonly angiocardiography fails to provide a complete clarification of the problem and cardiac catheterization is necessary as the ultimate procedure.

1. Defects in the Atrial Septum

An open foramen ovale is noted in 20 to 30 per cent of all necropsies. It has little clinical and practically no roentgenologic significance since a flap guards the opening, normally this prevents any blood from passing from the left atrium into the

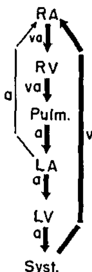


Fig. 181

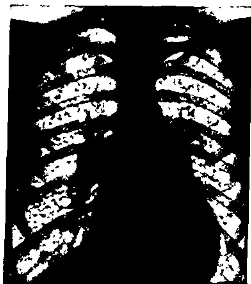


Fig. 182

Fig 181 —Atrial septal defect In all diagrams, a, v, av, indicate arterial, venous, and mixed blood, respectively. Arrows indicate direction of flow, thickness of line suggests volume

Fig 182 —Atrial septal defect. Male, 45 years old. Of graceful build and with moderate cyanosis and suggestion of watch-glass finger nails. Loud systolic murmur over heart with its maximum at Erb's point. Moderately enlarged cardiac shadow with elongation and marked *rounding of both borders and a protruding bulge of the pulmonary arc. The cardiac enlargement* was attributed to right ventricular hypertrophy and dilatation. The left atrium was not enlarged. The pulmonary arc and the dilated intrapulmonic branches of the pulmonary arteries showed very large pulsations. The aorta was narrow (arch diameter = 2.2 cm.).

right. Only a large atrial septal defect, irrespective of its origin from a maldeveloped septum primum or secundum or both, leads to cardiac alterations. While they occur as isolated arrests in development, in most cases (Abbott) they are associated with other anomalies or are accompanied by acquired lesions of an inflammatory nature.

Large defects in the interatrial septum, though isolated, can affect the heart profoundly, as Corvisart and Rokitansky noted. These alterations are the hemodynamic results of a quantity of blood passing from the left into the right atrium, consequently the right heart and lesser circuit are overfilled (fig 181). The right heart dilates from diastolic overload, its wall hypertrophies, augmented *filling and*

hypertension dilates the pulmonary artery and its branches while the poorly filled left ventricle remains relatively small or even atrophic. The aorta is narrow.

Clinically, the patient is usually pale The capacity for exertion is reduced and physical development somewhat retarded. Cyanosis is slight or absent The fingers are not clubbed Occasionally arrhythmias or paroxysmal tachycardias are observed. Cardiac dullness is enlarged. In the pulmonic area usually a systolic and sometimes a soft, blowing diastolic murmur is audible. The second pulmonic sound is accentuated and often palpable. Sometimes a systolic thrill is felt to the left of the sternum. Right ventricular hypertrophy and dilatation often produce distinct epigastric pulsations. Often this picture is complicated by a rheumatic valvulitis, especially of the mitral valve All these signs can be absent in childhood and they may appear only later There is a definite preference *for the female sex*

Cardiac catheterization yields the following a possibility of directly entering the left atrium through the septal defect, elevated pressure in the right atrium, the right ventricle and pulmonary artery, increased oxygenation of blood in the right atrium in contrast to that of the superior vena cava

The roentgen picture of a large, uncomplicated interatrial septal defect was originally described by Assmann and it corresponds thoroughly with hemodynamic conditions which might be anticipated (fig. 182). The more or less enlarged cardiac shadow has a mitral configuration and is enlarged predominantly to the left, there is massive bulging of the conus and the pulmonary artery Usually the pulmonary artery completely covers the aortic knob. The diameter of the aortic arch, providing it can be measured, is usually small.

Corresponding to the large stroke volume of the right ventricle, this chamber, and particularly the pulmonary arc, show striking pulsations.

The enlarged hilar shadows are surrounded by round, cherry-sized structures and broad linear markings, the round shadows are cross sections of the branches of the pulmonary artery caught end-on and the linear streaks are intrapulmonic branches cut lengthwise. The hilar and surrounding vascular shadows show strong systolic expansile pulsations. Otherwise the lungs are clear.

Examination in the left anterior oblique position confirms that cardiac enlargement depends upon increased volume of the right heart. In the right anterior oblique view, bulging of the pulmonary conus is particularly distinct while the left atrium is normal or only slightly enlarged, an important fact in differentiating mitral lesions

The prominence of the pulmonary arc and the enlarged shadows of pulmonary vessels resulting from dilatation of the pulmonary artery and its branches are particularly striking. The visible pulsations in these vessels exceed by far those observed *in mitral disease. Occasionally* they are further augmented by relative pulmonary insufficiency from marked dilatation of this valve orifice They are so striking even with the coexistence of a mitral lesion that a probable combination with an interatrial septal defect can be inferred from the remarkable pulsations of the pulmonary artery. Rarely, but particularly in young subjects, pulsations of the pulmonary arc and of the intrapulmonic vessels are less marked or absent

This roentgen picture permits only conjectures about the size of the interatrial septal defect It may be present with relatively small defects and absent with very large ones (Healey, Dow, Sosman and Dexter). In the latter instance pressure in the right heart is not elevated despite the demonstration of a left to right shunt by cardiac catheterization. In our experience, prominence of the pulmonary arc, while

often striking in adults, is missed fairly often in children The left to right shunt seems to increase as patients grow older; perhaps this depends upon a rheumatic lesion in the mitral valve or a gradual enlargement of the defect (Taussig). Moreover, Gefferth found merely a cardiac silhouette of nonspecific shape, rounded and bulging markedly to left and right, beneath a short vascular band in nurslings. At this age right atrial pressure can be greater than left and may lead to a right to left shunt, this opposes the dilatation of the pulmonary artery and occasionally leads to cyanosis (Taussig)

In a typical, pure atrial septal defect angiocardiography reveals enlargement of the right atrium and ventricle with more or less extreme dilatation of the pulmonary artery and its branches. The left atrium, normal in size, and the left ventricle

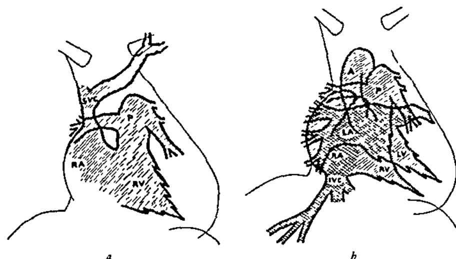


FIG 183.—Angiocardiogram of an atrial septal defect (a) Filling defect in the lower caval funnel during the injection of the contrast material; (b) reflux of blood containing opaque material into inferior vena cava and hepatic veins after refilling of right atrium through the interatrial septal defect.

opacify after the pulmonary circulation. Of diagnostic significance is the abnormally *prolonged opacification of the right heart owing to refilling of the right atrium via* the interatrial septal defect. In some cases the author made the following angiocardiographic observations. an initial filling defect in the right atrium (fig. 183a) and a belated reflux of opacified blood into the inferior vena cava (fig. 183b). The filling defect occurs in the region of the lower caval funnel with entry of the contrast material into the right atrium, on the contrary, opacification of the inferior vena cava and hepatic veins takes place only when blood containing opaque material is discharged from the lungs and enters the left atrium These two signs have some diagnostic significance and the obvious explanation is as follows: the blood stream shunted from the left to right atrium is directed toward the lower venous funnel, this deflects blood containing opaque material from the superior vena cava. On the other hand, opacified blood from the left atrium is propelled through the septal defect directly toward the veins which empty into the lower caval funnel.

When the interatrial septal defect is combined with emphysema or a congenital pulmonary stenosis (p. 279), right atrial pressure can be higher than left and a right to left shunt may be shown distinctly in the angiocardiogram. Healey and coworkers demonstrated a reversal from left to right shunt to right to left after exertion.

The combination of an interatrial septal defect and a mitral lesion, particularly mitral stenosis is common (Assmann, H. Muller Jr., Dressler and Rosler, Lutembacher, McGinn and White) It cannot always be decided whether the mitral orifice is narrowed by a postfetal endocarditis which developed on the basis of a congenital septal defect, from a congenital anomaly coordinated with the atrial septal defect as in a Duroziez mitral stenosis (Lutembacher), or from an acquired mitral stenosis



a *b*

FIG 184 —Large atrial septal defect with rheumatic mitral regurgitation and relative pulmonary and tricuspid insufficiency (necropsy) Underdeveloped female, 20 years old, with moderate cyanosis. No clubbing of fingers Systolic-diastolic murmur over the apex and pulmonary artery Ascites, congested liver Enormous cardiac shadow enlarged to left and right with massive protrusion of pulmonary arc and of left atrium which formed the right cardiac border Enormously enlarged hilar shadows Marked systolic-expansile pulsations on the pulmonary arc and of greatly dilated intrapulmonic arterial vessel shadows (*a*) Anterior view (*b*) left anterior oblique view.

in which heightened pressure in the left atrium caused dehiscence and gradual widening of the originally small foramen ovale.

The picture of a mitral lesion is modified essentially by a complicating atrial defect (fig. 184) Since blood from the left atrium can be routed into the right under higher pressure, too little reaches the left ventricle and systemic circulation while the right heart and pulmonic circulation are engorged. Accordingly, left atrial enlargement remains modest (Lutembacher) and the chamber may represent a mere appendage of the right On the contrary, the right atrium and ventricle are often enormous, the same affect acting on the pulmonary vessels is reflected in the massive enlargement of the hilar shadows and accentuated lung markings.

Usually these patients show only slight cyanosis and no clubbing of the fingers. Despite extreme mitral stenosis and decided cardiac enlargement, they may attain

often striking in adults, is missed fairly often in children. The left to right shunt seems to increase as patients grow older, perhaps this depends upon a rheumatic lesion in the mitral valve or a gradual enlargement of the defect (Taussig). Moreover, Gefferth found merely a cardiac silhouette of nonspecific shape, rounded and bulging markedly to left and right, beneath a short vascular band in nurslings At this age right atrial pressure can be greater than left and may lead to a right to left shunt, this opposes the dilatation of the pulmonary artery and occasionally leads to cyanosis (Taussig).

In a typical, pure atrial septal defect angiocardiography reveals enlargement of the right atrium and ventricle with more or less extreme dilatation of the pulmonary artery and its branches The left atrium, normal in size, and the left ventricle



FIG. 183.—Angiocardiogram of an atrial septal defect. (*a*) Filling defect in the lower caval funnel during the injection of the contrast material; (*b*) reflux of blood containing opaque material into inferior vena cava and hepatic veins after refilling of right atrium through the interatrial septal defect.

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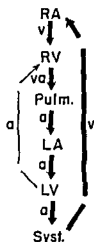


FIG. 185

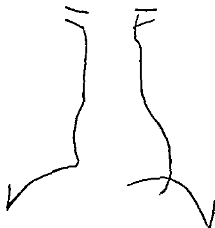


FIG. 186

FIG 185 —Ventricular septal defect

FIG 186.—Ventricular septal defect with normal radiographic findings Boy, 15 years old Dyspnea on running since childhood No cyanosis Jet-stream murmur over sternum.

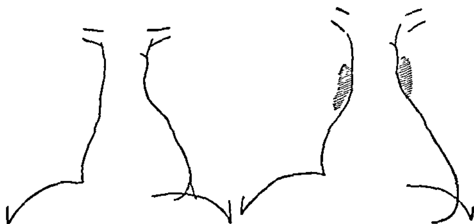


FIG 187 FIG 188

FIG 187 —Highly situated ventricular septal defect Normally developed female, 32 years old, without cyanosis Jet-stream murmur over sternum. The cardiac shadow, normal in size, showed a protrusion of the conus pulmonalis At this place marked systolic-expansile pulsations. The right cardiac border, descending laterad, showed large ventricular pulsations (Deneke's sign). The hilar shadows were not enlarged and showed no abnormal pulsations

FIG 188 —Large ventricular septal defect. Male, 20 years old Normally developed with slight cyanosis and suggestion of clubbed fingers Systolic murmur over heart with maximum at Erb's point Moderately enlarged cardiac shadow with elongated left ventricular arc, flat prominence of strongly pulsating conus pulmonalis and laterally descending, markedly bulging right cardiac arc which performed atrial pulsations The left atrium was not enlarged. The arterial shadows of the lung fields were wide but showed no increased pulsations

advanced age, some patients with this lesion have tolerated several pregnancies without apparent harm (Lutembacher).

Occasionally atrial septal defects morphologically resemble a high interventricular septal defect with an open ductus Botalli although cardiac enlargement as well as dilatation and pulsations in branches of the pulmonary artery are not as well developed. A similar situation is found also in Eisenmenger's complex accompanied by relative pulmonary regurgitation and in idiopathic dilatation of the pulmonary artery. In clinically doubtful cases *angiocardiography or cardiac catheterization* must be employed.

Pulmonary stenosis likewise leads to right ventricular hypertrophy and dilatation and frequently to marked dilatation of poststenotic section of the pulmonary trunk; it is distinguished from an atrial septal defect principally by the relatively small hilar shadows, the sparse vascular markings in the lungs, as well as by the absence of visible pulsations in the pulmonary vessels. The combination of an atrial septal defect and pulmonary stenosis is mentioned on page 283.

2. *Defects in the Ventricular Septum*

These defects usually occur in combination with and as a partial manifestation of torsion anomalies of the bulbus-truncus division, with or without stenosis of the pulmonary artery. An isolated interventricular septal defect is rare (Abbott, Laubry and Pezzi) *and will be discussed first. Usually it is located in the muscular part of* the septum and permits arterial blood to pass from the left ventricle into the right (Roger, Muller) (fig. 185).

[illegible]

develops in the pulmonary vessels, gas exchange is impaired (Taussig). In uncomplicated cases, cardiac dullness *is normal* or a *little enlarged. Over the sternum and in the left third or fourth* interspaces, usually a loud, prolonged murmur ("compressed jet stream murmur," Muller) is heard, frequently it is synchronous with a palpable purring thrill, occasionally a diastolic murmur is present. The second pulmonic sound is accentuated.

Cardiac catheterization reveals normal or somewhat elevated pressure in the right heart and greater oxygen saturation in the region of the outflow tract into which arterial blood enters from the left ventricle.

In many patients the heart is normal in size and shape (fig. 186). Fairly often, however, the pulmonary arc protrudes and the hilar and lung markings are accentuated (Muller, Assmann, Fuchs), pressure in the pulmonary arteries is elevated as the result of large amounts of blood entering through the left to right ventricular shunt (figs. 187 and 188). This happens particularly with defects located high in the ventricular septum when blood is forced through the defect into the outflow tract of the right ventricle or directly against the pulmonary valve. Sometimes the enlarged hilar shadows show strong systolic expansile pulsations. Occasionally the right cardiac border is rounded and descends laterally in the posteroanterior position; in the left anterior oblique position elongation and greater rounding of the anterior cardiac wall indicate hypertrophy and slight dilatation of the right ventricle. Moreover, sometimes the left ventricle is rounder, longer, and more *prominent owing to*

The roentgen findings are not remarkable. Usually enlargement of the right atrium and ventricle can be demonstrated. This finding, not striking in itself, the murmur, and the lowered oxygen saturation of arterial blood permit the probable diagnosis in a mongolian idiot (Taussig)

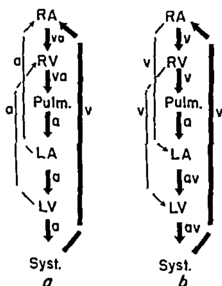


FIG. 189.—Persistent atrioventricularis communis (Canalis atrioventricularis communis persistens). (*a*) With left to right shunt, (*b*) with right to left shunt

4. Cor Triloculare Biatriatum by Complete Absence of the Interventricular Septum

In this malformation absence of an interventricular septum permits both atrioventricular ostia to open into a common ventricle From the outflow tract of this common ventricle which arises from the bulbus cordis, both vessels, or merely the pulmonary artery or aorta arise When the pulmonary artery alone arises from the conus-like outflow path of the common ventricle, its root lies in the same position as in the normal heart, that is, to the left and in front of the aortic root. On the other hand when the aorta arises from the conus-like section, it is located to the left and in front of the pulmonary root, then, the great arteries are transposed

Usually the artery originating from the conus-like outflow tract is more or less hypoplastic (Taussig). When this is the pulmonary artery, cyanosis is present because little blood is arterialized in the pulmonary circuit and most of the inadequately aerated ventricular content reaches the systemic circulation (fig 190a). With origin of the aorta from the conus section, cyanosis is absent because enough blood reaches the pulmonary circuit through the dilated pulmonary artery (fig. 190b), there is, however, some physical underdevelopment because the volume of blood in the general circulation is quantitatively inadequate despite its sufficient oxygenation (Taussig).

The relationships are, however, inconstant for there are many variants of this malformation Thus, Zdansky described a case of cor triloculare biatriatum in which

its hypertrophy and dilatation as the result of overloading from the pulmonary circulation In this way the cardiac shadow may become globular (Muir and Brown)

Powerful ventricular excursions of the right cardiac border, identical with those of the left ventricular arc were described by Deneke. Their occurrence is by no means regular and they are seen more frequently with large septal defects than with small ones (Thurn).

The roentgen picture of an interventricular septal defect sometimes recalls that of a persistent ductus arteriosus when the defect is located high in the septum so that the stream from the left ventricle is directed against the pulmonary ostium. In these cases pulmonary artery hypertension, as mentioned above, enlarges the hilar shadows and accentuates intrapulmonic vessels which show systolic expansile pulsations. A moderate bilateral cardiac enlargement completes the great similarity to a persistent ductus In a case of this type Morgan and Burchell noted, in addition to the systolic murmur, a diastolic one as well so that clinically one was also compelled at first to think of a persistent ductus Cardiac catheterization revealed the heightened oxygen saturation in the right ventricle indicating the existence of a ventricular septal defect, probably it was complicated by an aortic valve insufficiency of inflammatory origin

With small interventricular septal defects angiocardiography often proves uninformative (Dotter and Steinberg) Only when adequate amounts of blood pass from left to right through a large defect is persistent or even biphasic filling of the right ventricle detected, the latter occurs because it is refilled by blood from the left ventricle containing opaque material With high septal defects when blood from the left ventricle is ejected directly toward the pulmonary artery, one may occasionally observe biphasic filling of the pulmonary artery and its branches.

3. *Simultaneous Interauricular and Interventricular Septal Defects* (*Canalis Atrioventricularis Communis Persistens*)

When the formation of the atrial and ventricular septa remain incomplete, a large defect may develop which extends from the atria into the ventricles. Under these circumstances separation of the two atrioventricular ostia also fails to occur so that a single atrioventricular ostium represents the connection between the atria which are incompletely separated and the two ventricles which also intercommunicate.

Since the pressure in the left atrium and ventricle usually exceeds that in corresponding divisions of the right heart, at first blood passes from the left heart to the right (fig. 189a) This raises pressure in the right heart until this is higher than in the left, then, the flow in the shunt is reversed (fig. 189b) until pressure on the left heart rises above that in the right. Consequently bidirectional changes of flow in the shunt tend to recur (Taussig). In the course of years the heart progressively enlarges although the increase remains within modest limits and involves mainly the right atrium and ventricle.

This arrest in development is particularly common in mongolian idiots (Taussig). There is a rough systolic murmur and thrill over the heart just as with an interventricular septal defect. Cyanosis and clubbed fingers do not belong to the clinical picture of this malformation although oxygen saturation in the arterial blood tends to remain low.

a case with atresia of the pulmonary artery and persistent ductus arteriosus. In other words, numerous variants and combinations of this malformation occur.

These remarks indicate that the clinical findings are variegated In the early months of life no murmurs are heard Gradually a systolic and sometimes a presystolic murmur become audible. The systolic murmur is not loud or rough as in Fallot's anomaly but is soft

Cardiac catheterization reveals increased oxygen saturation in the ventricle in contrast to that of the right atrium. Within the ventricle whose pressure is elevated, oxygen saturation varies according to the relation of the catheter orifice to the site of inflow of the right or left atrium The oxygen saturation in the ventricle is approximately the same as in the femoral artery

Despite the anatomic differences the roentgen picture is relatively uniform There is a more or less distinct projection above the left ventricular arc where a conus pulmonalis protrudes when dilated. This corresponds to the conus-like section of the single ventricle from which either one or both of the great arteries arise (Fig 191a, b). Naturally this is less prominent when the diaphragm is low In cyanotic patients the pulmonary vascular markings are diminished, in acyanotic ones they are increased and even pulsate. A suspicion of right ventricular hypertrophy and dilatation might be awakened on the basis of the conus-like prominence on the left cardiac border, but, in the left anterior oblique position the anterior wall of the heart descends sharply and flatly to the diaphragm, an important point in differential diagnosis.

Angiocardiography provides information about particular features in single cases. It permits one to recognize the common ventricle and simultaneous filling of the pulmonary artery and aorta The reciprocal positional relations and the diameters of the two great arteries as well as the development of the brachiocephalic branches of the aorta may be clarified

Cyanotic cases can be definitely helped by the Blalock-Taussig operation

5. Anomalies of the Tricuspid Ostium

Tricuspid atresia. Tricuspid atresia appears in two forms 1) "Isolated" tricuspid atresia There is an associated hypoplasia of the right ventricle and a ventricular septal defect or an aplasia of the right ventricle and persistent ductus arteriosus 2) Tricuspid atresia with transposed vessels

Exceptionally the right ventricle develops as a small blind sac devoid of function and connected with the right atrium through a very narrow atrioventricular valve. Necessarily, there is a persistent ductus arteriosus. All forms of tricuspid atresia have a common factor in that blood entering the heart can pass into the left half only through an atrial defect.

In "isolated" tricuspid atresia the blood is invariably discharged through an interatrial septal defect into the left atrium and hence through the mitral orifice into the left ventricle and aorta When a hypoplastic right ventricle is present, a small quantity of blood is pushed through an interventricular septal defect into this small right ventricle, which functionally is but a junction with the hypoplastic pulmonary artery (fig. 192a). The dominant manifestations of this anomaly, tricuspid atresia, atrial and ventricular septal defects are summarized as Wieland's triad. When the right ventricle fails to develop completely the sole possibility for supplying the

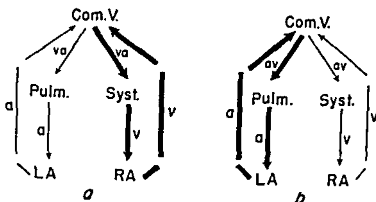


Fig 190.—Cor triloculare biatriatum. (*a*) with narrow pulmonary artery, (*b*) with narrow aorta.

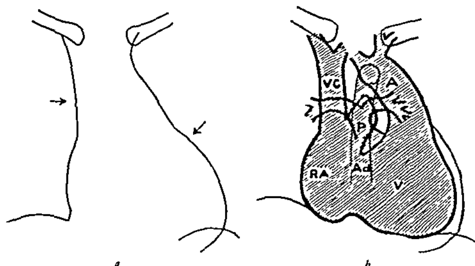
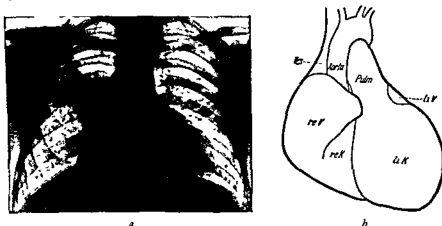


Fig. 191 —Cor triloculare biatriatum with transposition of great vessels Underdeveloped boy, 9 years old, with severe cyanosis since birth and clubbed fingers. Erythrocytes, 6 7 millions. Soft systolic murmur over the heart with point of maximum intensity over the pulmonic Accentuation of the second pulmonic sound Blood pressure 90/55 mm. Hg. Electrocardiogram right preponderance. (*a*) Heart (erect position) The cardiac shadow is enlarged a little with slight prominence in the cardiac waist and at the cephalic limit on the left cardiac border as a sort of conus (arrow). The right border of the vascular band descends perpendicularly (arrow) since the ascending aorta ascends in the left mediastinum. The hilar shadows are small (*b*) Angiocardiogram (recumbent position) simultaneous opacification of the aorta and narrow pulmonary artery from a common ventricular chamber The vessels are transposed. Successfully operated according to the Blalock-Taussig procedure

the aorta arose to the left and in front of the pulmonary artery from the conus of the solitary ventricle owing to transposition of the great vessels The aorta was wide and the pulmonary artery narrow (fig 191a, b). Since most of the inadequately aerated blood from the ventricle was advanced into the systemic circulation, there was deep cyanosis and clubbed fingers. Over the heart, there was a low, soft systolic murmur. Apparently this type is not extremely uncommon for Donzelot and coworkers as well as Thurnher and Weissel published similar cases. Taussig described

In tricuspid atresia with transposed arteries, blood of the right atrium passes through an interatrial septal defect into the left half of the heart, the abnormally wide pulmonary artery arises from the dilated and hypertrophied left ventricle at the left behind the aorta, the aorta, usually narrow, arises from the hypoplastic right ventricle to the right and in front of the pulmonary artery, the right ventricle obtains its blood through an interventricular septal defect from the left ventricle (figs. 192c and 193a and b). Sometimes the ventricular septum is entirely absent and one large ventricle gives rise to the wide pulmonary artery and the narrow aorta (fig. 192d). The aorta on the left and in front of the pulmonary artery arises from a pouch in the single "aortic" ventricle. In other words an anatomic cor triloculare biatriatum is present

If the great arteries are transposed, venous blood enters the left half of the heart through the interatrial septal defect from which it is discharged directly into the



a b

Fig. 193.—Tricuspid atresia with transposition of aorta and pulmonary artery, atrial and ventricular septal defect. Male 20 years old Cyanosis of variable intensity. Death from cavernous tuberculosis See text for description Vcs, superior vena cava, ReV, right atrium, liV, left atrium, reK, right ventricle, liK, left ventricle.

pulmonary artery arising from the left ventricle and into the pulmonary circulation. Consequently the left ventricle receives venous blood from the systemic circulation and arterialized blood from the lungs The oxygen content of the mixed blood determines whether or not cyanosis will appear and its depth. At all events the situation is definitely more favorable than in "isolated" tricuspid atresia; this is shown by the greater average duration of life (9 3 years). The most advanced age attained was 56 years (Hackensellner).

Taussig mentions atresia of the transposed pulmonary artery in which the rudimentary right ventricle obtained its blood via an interventricular septal defect while the systemic as well as the pulmonary circulation were supplied by the aorta arising from the right ventricle and a persistent ductus.

At times tricuspid atresia is combined with dextrocardia (Wittenborg, Neuhauser, and Sprunt). Frequently an arcus aortae dexter is also present.

A cardiac catheter as a rule passes out of the right into the left atrium so that right-sided venous blood and left-sided mixed blood is obtained From the left atrium one may advance to the left

pulmonary circulation is a persistent ductus arteriosus and ectatic bronchial arteries (fig 192b).

At all events the left ventricle is entirely responsible for propelling blood from the heart.

Despite differences in the circulatory situation in the two types, the oxygen supply of the organism is adversely affected in both so that cyanosis results. Corresponding to hypoplasia or complete

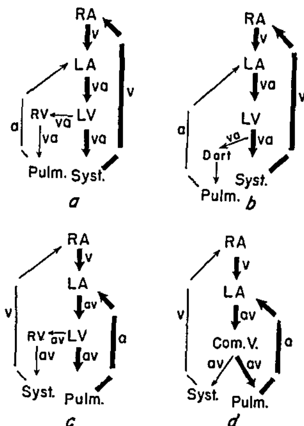


FIG. 192 —Tricuspid atresia (a) Isolated tricuspid atresia with hypoplasia of right ventricle and interventricular septal defect, (b) isolated tricuspid atresia with aplasia of the right ventricle and persistent ductus arteriosus, (c) tricuspid atresia with transposition of the great vessels, hypoplasia of the right ventricle and interventricular septal defect, (d) Tricuspid atresia with transposition of the great vessels and common ventricle

which has right preponderance On the other hand one must not forget that left preponderance and cyanosis may be combined in cases of atrial septal defect with pulmonary stenosis, in cases of cor triloculare biatriatum and in cases of transposition of the great vessels with pulmonary stenosis (Wittenberg, Neuhauser, and Sprunt) Death usually occurs in the first year (average survival 1.7 years). Survival lengthens with the size of the atrial defect, with a persistent ductus arteriosus and ectatic bronchial arteries, and when a hypoplastic right ventricle communicates with the left through a septal defect. The oldest age attained is reported as 27 years (Hackensellner).

the ventral position of the aorta, visible in oblique positions; 2) in the enlarged hilar shadows and accentuated markings of vessels in the lungs which may exhibit systolic expansile pulsations. If a large solitary ventricle is present owing to nondevelopment of the ventricular septum, then the flat, sharp descent of the anterior cardiac

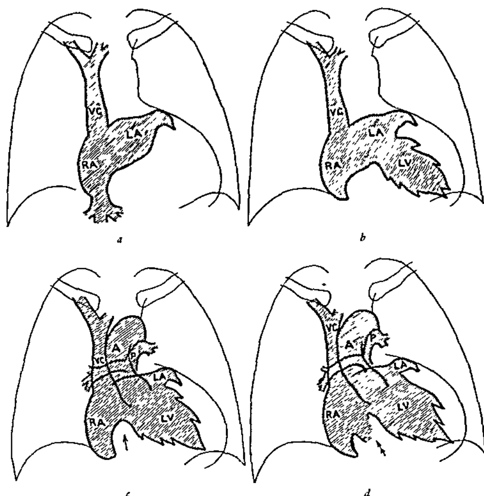


FIG 195 —Angiocardiogram in tricuspid atresia. (*a*) Opacification of left atrium by an atrial septal defect (*b*) Opacification of the left ventricle from left atrium, Filling defect between right atrium and left ventricle (*c*) Opacification of the aorta from left ventricle and pulmonary artery through persistent ductus arteriosus. Filling defect the result of aplasia of the right ventricle unchanged (arrow) (*d*) Opacification of aorta from left ventricle and pulmonary artery through hypoplastic right ventricle which communicated with the left ventricle through a septal defect. Reduction of the filling defect by opacification of small right ventricle.

wall is absent Zdansky observed a pertinent case. A somewhat underdeveloped 20 year old male had exhibited cyanosis of variable depth from early childhood; physically he had been much below par. The erythrocytes numbered 6.25 millions. A loud systolic murmur and a palpable thrill were present over the heart. The second

ventricle and, with normal situs of the great vessels, to the aorta. If an interventricular septal defect is present, occasionally one also enters the pulmonary artery. If the great vessels are transposed, one may enter the pulmonary artery from the left ventricle.

In "isolated" tricuspid atresia usually the cardiac shadow expands a little to the left and the left cardiac border is more definitely rounded, the apex is elevated somewhat. In some cases absence or hypoplasia of the right ventricle can be recognized by the failure of the right cardiac border to project over the vertebral column, it descends flatly and sharply (fig. 194) sometimes even laterally and concavely within the spinal shadow to the diaphragm. In most cases, however, the findings are less characteristic and the right cardiac border is about normally rotund. Since the apex may also seem somewhat elevated the picture of *coeur en sabot* may develop (Neuhauser) Pictorally, such cases are not distinguishable from a Fallot anomaly In both, the aortic arch fairly often crosses the right bronchus. The hilar shadows are

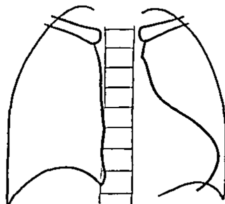


FIG 194 —Tricuspid atresia with perpendicular and flatly descending right cardiac border which just reaches the shadow of the spine Coeur en sabot

abnormally small. Sometimes widened bronchial arteries pass from the mediastinum into the lung fields.

The angiocardiogram has decisive diagnostic significance. Naturally, reliable conclusions about the characteristic process of filling are secured only with films obtained in rapid sequence like a moving picture, the decisive phases unfold within one or two seconds (Janker and Hallerbach). Then, opaque material immediately passes from the right into the left atrium and to the dilated left ventricle (fig 195a and b). At the site of the atretic or hypoplastic right ventricle, between the visualized right atrium and left ventricle, there is a defect in the shadow. After the left ventricle is opacified, the aorta is visualized. In patients with a complete atresia of the right ventricle the defect in contrast filling may be marked (fig 195c) The pulmonary artery becomes filled belatedly from the aorta through the persistent ductus arteriosus. In patients with a ventricular septal defect, the defect in the shadow is less; sometimes a small right ventricular space may be visualized (fig 195d) as well as almost simultaneous filling of the aorta and pulmonary artery which arise from the hypoplastic right ventricle (Janker and Hallerbach).

When tricuspid atresia is associated with transposition of the great vessels, the picture differs from that of isolated tricuspid atresia in the following respects: 1) in

the ventral position of the aorta, visible in oblique positions, 2) in the enlarged hilar shadows and accentuated markings of vessels in the lungs which may exhibit systolic expansile pulsations If a large solitary ventricle is present owing to nondevelopment of the ventricular septum, then the flat, sharp descent of the anterior cardiac

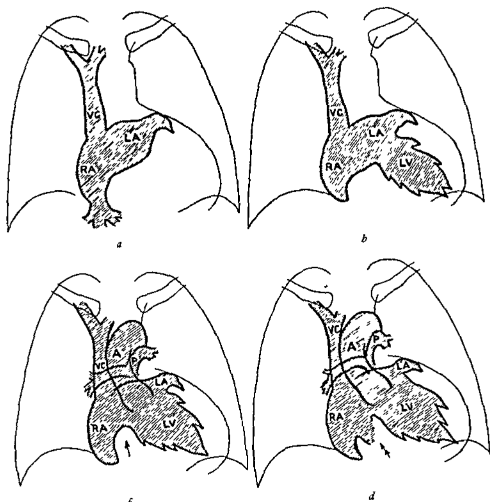


FIG 195 —Angiocardiogram in tricuspid atresia (*a*) Opacification of left atrium by an atrial septal defect. (*b*) Opacification of the left ventricle from left atrium, Filling defect between right atrium and left ventricle (*c*) Opacification of the aorta from left ventricle and pulmonary artery through persistent ductus arteriosus. Filling defect the result of aplasia of the right ventricle unchanged (arrow) (*d*) Opacification of aorta from left ventricle and pulmonary artery through hypoplastic right ventricle which communicated with the left ventricle through a septal defect. Reduction of the filling defect by opacification of small right ventricle.

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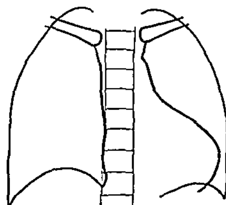


FIG 194 —Tricuspid atresia with perpendicular and flatly descending right cardiac border which just reaches the shadow of the spine Cocur en sabot.

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When tricuspid atresia is associated with transposition of the great vessels, the picture differs from that of isolated tricuspid atresia in the following respects: 1) in

the ventricle preserves only its apical section and the outflow tract while most of the inflow tract functionally is drawn into the atrium *Usually the valves are incompetent.* No abnormal murmurs or cyanosis are present, but extrasystoles and paroxysmal tachycardias occur.

The right atrium, enlarged at the cost of the right ventricle, shows nothing characteristic. Consequently the clinical diagnosis of this anomaly is uncertain. Taussig observed universal enlargement of a heart, originally *normal in size and* shape over a period of six years, the young man suffered continuously from paroxysmal tachycardia and intraventricular block which proved fatal.

By angiocardiography one may expect to see displacement of the tricuspid valve toward the apex with a large right atrium and a small right ventricle.

6. *Anomalies at the Base of the Pulmonary Artery*

Congenital pure pulmonary stenosis In a vast majority of cases pulmonary stenosis is a feature of complicated malformations such as Fallot's anomaly (p 298) or hypoplasia of the right ventricle (p. 274) Pulmonary stenosis either pure or associated with an atrial septal defect is usually located at the pulmonary ostium for the valves are formed imperfectly or adhere more or less to one another More rarely the stenosis is infundibular, that is, in the initial part of the conus pulmonalis, in the entire infundibulum, in the trunk of the pulmonary artery or, finally in the pulmonary ostium and adjoining parts

Irrespective of its site, the increased resistance caused by the stenosis always leads to dilatation and hypertrophy of the right ventricle although this may be trifling for a long time. Furthermore, the section distal to the stenosis tends to dilate. If the stenosis is located at the infundibular ostium, the conus and pulmonary artery dilate, with stenosis of the infundibulum or pulmonary valve, the dilatation of the pulmonary artery may extend down to its division into the two major branches The dilatation distal to the stenosis may be considered an anatomic fixation of the dynamic dilatation Volhard explains the latter by the impact of blood forcibly ejected from the hypertrophied right ventricle

Pure pulmonary stenosis causes no complaints as long as the right ventricle can surmount the heightened resistance by means of hypertrophy and dilatation If the stenosis is slight this may last for decades If, however, the stenosis is marked, dilatation due to incompetence *of the right ventricle often appears early and finally leads to signs of failure (dyspnea, cyanosis, passive congestion* in the systemic circulation) The average expectation of life is about 22 years (Abbott)

Over the pulmonic area in the second and third intercostal spaces, just to the left of the sternum and corresponding to a palpable thrill, a loud, rough systolic murmur is audible, it is conducted upward and into the back Usually the second pulmonic sound is weak but sometimes it is accentuated There is dullness in the region of the cardiac waist but cardiac enlargement is not invariable Distinct ventricular pulsations may be seen and felt in the epigastrium

In the compensated stage of pure stenosis, cardiac catheterization reveals normal pressure in the right atrium, elevated pressure in the right ventricle, and low pressure in the pulmonary artery Oxygen saturation in the right heart and in the pulmonary artery is normal in the stage of compensation

In well compensated, low grade stenosis, the cardiac shadow is normal in size and not striking in shape Usually, however, a mitral configuration exists owing to prominence of the pulmonary arc (fig. 196) resulting from dilatation of the conus

pulmonic sound was accentuated. Diabetes mellitus was present. Death resulted from caseous pneumonia. The moderately enlarged heart had an aortic configuration and was widened mainly to the left; the rounded left cardiac border projected markedly and the aortic knob was absent. The diameter of the aortic arch was not determined. The hilar shadows were somewhat enlarged but showed no striking pulsations In the middle and basal parts of the right lung, his tuberculosis created mottling. Autopsy revealed an atresia of the tricuspid valve with atrial and ventricular septal defects, both of which admitted a finger. Both atria were enlarged. The large hypertrophied left ventricle gave off the pulmonary artery and the very small right ventricle the narrow aorta (fig. 193a and b).

The angiocardiogram permits immediate recognition of transposition of the great vessels since the dilated pulmonary artery is immediately filled from the left ventricle, the contrast substance has passed through the atrial septal defect into the left heart. In most cases the intrapulmonic branches of the pulmonary artery are *distinctly dilated. The hypoplastic aorta lies in front of the pulmonary artery* and fills faintly. Since the ventricular septum is absent, only one large ventricular space is present and the shadow of the right heart is not defective; consequently the tricuspid atresia is demonstrable only when the sequence of filling is followed exactly by films at short intervals.

Decisions on the indications for a Blalock-Taussig operation are aided greatly by angiocardiography. The operation comes under consideration only with isolated tricuspid atresia and is contraindicated with transposition of the vessels.

Congenital tricuspid regurgitation. Congenital tricuspid insufficiency is rare. Hotz described two children, aged 12 and 13 years, who had suffered from cyanosis and dyspnea from earliest childhood. Both developed cardiac failure with increasing cardiac enlargement which proved fatal within a short time. The hearts were enlarged mainly to the left and the second pulmonic sound was definitely accentuated, one case had a variable soft systolic murmur over the pulmonary artery, the other had a positive venous and hepatic pulse terminally.

In both cases the cardiac shadow had a mitral configuration, the bulging pulmonary arc completely filling the cardiac waist. The hearts enlarged progressively and expanded mainly to the left The right cardiac border displayed augmented pulsations. Pulmonary congestion was absent. Necropsy revealed that the cardiac enlargement depended exclusively upon the right atrium and ventricle and the tricuspid valve admitted three to four fingers. In both patients the trunk of the pulmonary artery was markedly dilated and the aorta was narrow. The ductus arteriosus was closed.

Apart from the existence of the lesion since earliest childhood, its congenital nature was further attested by the fact that in both cases the tricuspid valves were anomalous in a manner which could not have been acquired. In one patient only two leaflets were present and in the other a normal leaflet was associated with two rudimentary ones. Hotz interpreted the remarkable width of the pulmonary artery as the result of unequal division of the truncus

Ebstein's disease. This rare anomaly is characterized by the fact that the tricuspid valve does not arise from the annulus fibrosus but is partly displaced into the ventricle so that part of the latter is reduced and incorporated into the atrium. Accordingly,

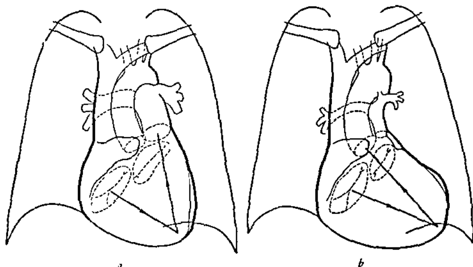


Fig 197 —Course of the inflow and outflow tracts of the right ventricle in mitral stenosis (a) and in pulmonary stenosis (b) with ventricular septal defect and riding aorta (schematic). In the first instance the lengthening of the outflow tract produces a mitral configuration owing to its nearly perpendicular course while in the second instance, owing to its oblique course, coeur en sabot ("wooden shoe heart") appears The narrowness of the conus and the pulmonary artery still contribute definitely to the preservation of the cardiac waist in pulmonary stenosis

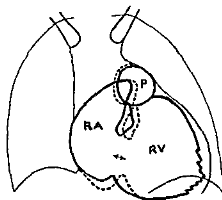


Fig 198 —Dextrogram of a congenital pulmonary stenosis The alterations of volume of the right atrium and right ventricle are very slight During several cardiac cycles there was no definite emptying of the right heart Owing to the relative tricuspid regurgitation, the opacified blood ran back and forth between atrium and ventricle ——— diastolic, ----- systolic

films in diastole permit reliable conclusions on the width of the infundibulum (p. 15)

The electrokymogram may permit one to differentiate a valvular from an infundibular stenosis (Andersson). In both instances the systolic limb of the pulmonic curve ascends more slowly than normal, in a valvular stenosis, however, the initial oblique rise with a subsequent notch is followed by a relatively flat ascent to the peak of the curve, in an infundibular stenosis the systolic limb of the curve climbs

and the pulmonary artery (Vaquez and Bordet, Burke, Assmann, Dressler, Usomoto, Eakin and Abbott).

The exact site of the stenosis within the bulbus-truncus section is not perceptible. In striking contrast to this dilatation of the pulmonary artery are the small hilar shadows as well as the clear lung fields which show a poverty of markings. This is important in differentiating large atrial septal defects, Eisenmenger's complex (p 307), open ductus Botalli, cor pulmonale from primary pulmonary sclerosis, high grade emphysema, or mitral lesions. Pulmonary stenosis is further distinguished from the last by the absence of left atrial enlargement.

While the cardiac shadow may be normal in size, sometimes right ventricular dilatation enlarges the shadow considerably The expansion is mainly to the left as long as the right atrium does not dilate from right ventricular failure and retrograde



FIG 196—Congenital pulmonary stenosis Female, 35 years old, with mild exertional dyspnea Slight cyanosis, no clubbing of fingers, loud rough systolic murmur over the heart with maximum over the pulmonic area, transmitted to the back Absent second pulmonic sound Plump cardiac shadow widened to right with massive protrusion of pulmonary arc behind the aortic knob which has almost vanished No increased pulsations on pulmonic arc Left atrium and ventricle not demonstrably enlarged Hilar shadows normal in size and without striking pulsations Lung fields clear

stasis. In the latter instance the cardiac shadow of mitral configuration also expands to the right with an elongated and laterally descending arc projecting into the lung field

Obviously the roentgen picture of a pure pulmonary stenosis differs basically from pulmonary stenosis in a Fallot anomaly. The reasons for the difference are given on page 301 (fig. 197) Angiocardiography and roentgenkymography supplement the roentgen findings since they yield valuable information on the site and extent of the stenosis; this may be important in decisions on the type of surgery contemplated.

Angiocardiography reveals the degree of dilatation of the right atrium and ventricle. With decompensation often the right heart empties very slowly. With relative tricuspid insufficiency serial films show how the right heart blood, almost stagnant, moves to
and extent of stenos
connection attention
may contract so strongly in systole than an infundibular stenosis is simulated. Only

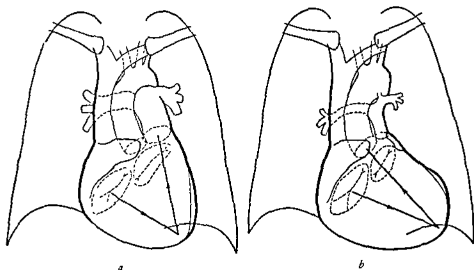


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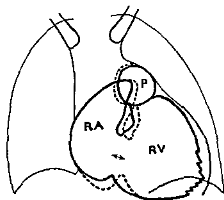


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FIG 199 —Angiocardiogram of a congenital pulmonary stenosis located at the infundibulum

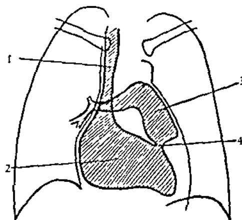


FIG 200 —Angiocardiogram of a congenital conus stenosis of the right ventricle (After Sussman and Grishman)

1 V cava sup.
2. Right heart
3. Art pulmonalis
4 Stenosis of the conus pulmonalis

gradually so that a sinuous curve develops. The electrokymogram becomes normal when valvulotomy relieves a pure valvular stenosis or it assumes a sinuous form when an infundibular stenosis remains (Andersson).

Rarely, complete atresia of the pulmonary artery leaves the right ventricle without an exit so that cardiac blood is discharged exclusively through the aorta which arises from the left ventricle. This atresia is incompatible with life without an atrial

septal defect which permits venous blood to pass from the right into the left atrium, and a persistent ductus arteriosus by which part of the mixed blood of the left heart may pass into the pulmonary circuit for aeration. The maximum duration of life with this malformation was six months (Abbott). Kugel reported the roentgen findings (fig. 201). There was enormous enlargement of the right atrium and ventricle, the former showed great hypertrophy and the latter was very thin. The thinness and dilatation of the right ventricle which ended blindly was ascribed to improper coronary perfusion. The moderately hypertrophied left ventricle gave rise to a trivalvular aorta. An enormous cardiac shadow bulged to the left and even more to the right below a very short vascular band. The right cardiac border was very striking, it began just below the clavicle and descended to the diaphragm in a huge arc formed by the large right atrium and ventricle.



Fig. 201.—Atresia of pulmonary artery (case of M. A. Kugel). An infant of 6 months with severe cyanosis and clubbed fingers. Rough systolic murmur with maximum intensity in the second left intercostal space. Large liver (necropsy).

Congenital pulmonary stenosis with atrial septal defect. The combination of pulmonary stenosis with defective atrial septum, the "trilogy of Fallot" is a little more common than pure pulmonary stenosis. This combination represents an unloading of the right heart since blood is able to pass from the right into the left atrium, on the other hand, this allows venous blood to reach the left heart and systemic circulation (fig. 202). If the septal defect is large and the pulmonary stenosis relatively marked, moderate cyanosis and dyspnea may occur. The stenosis is located more often at the valves than in the infundibulum.

The auscultatory findings are like those of a pure pulmonary stenosis.

Cardiac catheterization shows a difference from pure pulmonary stenosis since the left atrium may be entered through the septal defect and since pressure in the right ventricle is less elevated.

While the roentgen picture resembles pure pulmonary stenosis in many respects, the left heart may appear somewhat enlarged owing to the right to left shunt.

Angiocardiography distinctly visualizes the enlarged right atrium and ventricle as well as the poststenotic dilatation of the pulmonary artery. Moreover, the position and extent of the stenosis frequently can be defined. Almost simultaneously with the right heart and the pulmonary artery appears a faint opacification of the left atrium, the left ventricle and the aorta by way of the interatrial septal defect. With a large atrial septal defect sometimes the left atrium and ventricle may become

opaque earlier than the right ventricle. Then, opacification of the aorta may antedate visualization of the pulmonary artery (Zdansky). Naturally the belatedly filled right ventricle and poststenotic pulmonary artery may remain opaque long

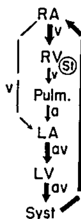


Fig. 202

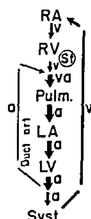


Fig. 203

Fig. 202 —Pulmonary stenosis with interatrial septal defect

Fig 203 —Pulmonary stenosis with persistent ductus arteriosus

after the left heart and aorta have faded. On the other hand the latter becomes reopacified when the contrast substance has passed the pulmonary circulation This biphasic levogram is most distinct when a relatively small amount of the contrast substance is injected rapidly and views are obtained in rapid sequence (Holzmann).



Fig. 204.—Congenital anomaly, probably pulmonary stenosis with persistent ductus Botalli Female, 28 years old. See text for description Unfortunately the dilatation of the left pulmonary artery is not very clear on the figure

Congenital pulmonary stenosis with persistent ductus arteriosus. Zdansky described three cases in which the roentgenologic and clinical findings suggested a combination of congenital pulmonary stenosis and persistent ductus arteriosus (fig. 203). Figure 204 shows the roentgenogram of one case. The cardiac lesion of this 28 year

old woman had been discovered in early childhood She suffered from slight dyspnea after strenuous exertion but had neither cyanosis nor clubbed fingers. A loud, prolonged, systolic murmur was audible over the entire heart, its maximum intensity was over the second and third left intercostal spaces where it was accompanied by a palpable thrill The murmur was transmitted to the lungs and back. The second pulmonic sound was absent. Peripheral pulsations were normal.

The cardiac shadow was moderately enlarged, its elongated and markedly rounded right border showed ventricular pulsations. A prominent pulmonary arc with systolic expansile pulsations of remarkable size filled the cardiac waist. Identical pulsations were seen in the left pulmonary artery which was extraordinarily dilated while the right pulmonary artery, normal in caliber, did not show them Pulmonary congestion was absent. The left atrium was normal in size Identical findings in the three cases of Zdansky, none of whom had noteworthy circulatory complaints seemed to favor a typical anomaly Necropsy and angiocardiographic and cardiac catheterization control were not possible but the absence of definite circulatory disturbances, the presence of a rough systolic murmur over the pulmonic area, absence of the second pulmonic sound, as well as a hypertrophied right heart with prominence of the pulmonic trunk spoke in favor of pulmonary stenosis On the other hand, the large pulsations of the pulmonary trunk and the isolated dilatation of the left pulmonary artery made a persistent ductus arteriosus probable. The occasional concurrence of persistent ductus arteriosus and pulmonary stenosis is mentioned by Grohs, Burchell, Taussig, and others

The diagnosis is established by cardiac catheterization. Pressure is somewhat elevated in the right ventricle and lowered in the pulmonary artery, venous oxygen saturation in the right atrium and ventricle as well as increased oxygen tension in the pulmonary artery are noted

While Taussig opposes ligation of a ductus arteriosus in the presence of a pulmonary stenosis, others (Taylor and Du Shane) are less opposed, particularly when danger of subacute bacterial endocarditis exists.

Pure congenital pulmonary regurgitation. This rare malformation, often associated *with supernumerary or an insufficient number of valve leaflets, is combined with* right ventricular hypertrophy and dilatation in correspondence with the dynamics of the lesion, the other cardiac chambers suffer no alterations until the right ventricle begins to fail The trunk of the pulmonary artery and its branches tend to dilate.

The *moderately enlarged heart (Kautsky, Kissin) has a mitral configuration* and is widened mainly to the left. The cardiac waist is completely filled by the protruding pulmonary arc. The hilar shadows and vascular markings of the lungs are unusually large. The cardiac shadow shows large pulsations especially on its right border reflecting the augmented right ventricular *stroke volume The pulmonary arc*, hilar shadows and the arterial branches show excursions like a pulsus celer The picture is not characteristic. Fundamentally, it is not even distinguished from the equally rare, pure acquired organic pulmonary insufficiency, from large atrial septal defects, and at times, from patent ductus arteriosus Only its congenital nature and a diastolic murmur of regurgitation over the pulmonic valve make the diagnosis probable. One must, however remember that relative valvular incompetence may merely accompany or result from an atrial septal defect. The presence of excursions

like pulsus celer in the mainstem of the pulmonary artery and its dilated branches differentiates it from a pure congenital pulmonary stenosis.

7. Anomalies at or Adjoining the Aortic Valve

Pure congenital aortic stenosis. Congenital pure aortic stenosis is one-tenth as common as congenital pure pulmonary stenosis (Rauchfuchs). It can also involve the conus arteriosus or the valve. If valvular, it is often uncertain whether the stenosis is congenital or inflammatory in origin Conus stenosis (subaortic stenosis) usually lies about 1 cm below the valve as a diaphragm-like membrane, it is always a malformation. Valvular stenosis shows no preference for either sex while conus stenosis favors the male

The hemodynamics correspond thoroughly with those of postinflammatory aortic stenosis Consequently the results, demonstrable clinically and roentgenologically, are the same (Muller, Jr, Mannheimer and Nordenfelt, Taussig, and others). Although sudden death or superimposed endocarditis may occur, the prognosis is relatively favorable unless the stenosis is extreme. Cyanosis is absent and the physical capacity of the patient is affected little or not at all.

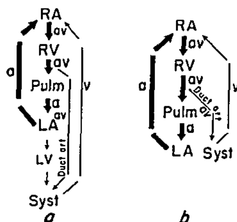


Fig. 205 —Congenital aortic stenosis (a) With hypoplasia of the left ventricle, (b) with aplasia of the left ventricle and persistent ductus arteriosus

Cardiac catheterization reveals nothing abnormal.

The presence of signs since infancy, the absence of a previous endocarditis, the existence of a stenosis prior to the appearance of an aortic valve regurgitation speaks in favor of the congenital origin (Taussig)

dilatation

Atresia or hypoplasia of the aorta with underdevelopment of the left ventricle. Congenital hypoplasia of the aorta which may be associated with complete occlusion of its ostium, is accompanied by underdevelopment of the left ventricle and stenosis or atresia of the mitral valve The hypoplasia extends upward to the orifice of the ductus arteriosus which is always patent With atresia of the aortic valve the hypo-

plastic aorta must always receive its blood by retrograde flow from the ductus arteriosus Regularly an atrial septal defect is present through which blood passes from the left atrium into the right (fig. 205a, b). This defect is largest when the left ventricle is entirely aplastic and smaller when it is hypoplastic. With aplasia of the left ventricle the aerated blood which the left atrium receives from the lungs passes through the interatrial septal defect into the right heart (fig. 205b). The right ventricle pumps mixed blood through the pulmonary artery to the lungs as well as to the systemic circulation through the patent ductus arteriosus. If the left ventricle is hypoplastic, some blood from the left atrium may enter the hypoplastic aorta through the small left ventricle (fig. 205a). In both instances the chief task of propulsion falls on the right ventricle which undergoes extreme dilatation owing to diastolic overloading and systolic resistance acting on its thin wall This is present even at birth since the right heart is abnormally burdened even during fetal circulation There is always extreme cyanosis and dyspnea, low peripheral blood pressure, and a congested liver (Taussig) Usually the anomaly proves fatal shortly after birth.

Corresponding to the left to right shunt, cardiac catheterization reveals considerable elevation of pressure and heightened oxygen tension in the right atrium and ventricle From the pulmonary artery which also stands under high pressure, one may occasionally pass through the ductus arteriosus into the aorta where the oxygen tension is the same or not essentially different from that in the pulmonary artery

Taussig reported two roentgen findings. Even in the first days of life, the cardiac shadow was enormously enlarged and in one case mainly to the right. Corresponding to the right ventricular hypertrophy and dilatation, the conus pulmonalis is prominent In the left anterior oblique position the anterior wall of the heart extended to the chest wall The shadow of the superior vena cava was widened Zdansky has never seen such a case

Congenital aneurysm of the sinus of Valsalva This malformation is discussed with aneurysms of the aorta (p 392)

8 Truncus and Pseudotruncus Arteriosus Communis

Truncus arteriosus communis is characterized by a single, abnormally wide vessel arising over a ventricular septal defect to carry blood from both ventricles to the lungs as well as to the systemic circulation. The coronary arteries are also supplied by this vessel

Two different although functionally similar forms are distinguished a) true truncus arteriosus communis results from the failure of a septum to form and divide the truncus, b) pseudotruncus arteriosus communis occurs when the septum divides the truncus unequally so that the antimere which corresponds to the pulmonary artery becomes an atretic cord Pseudotruncus is much more common than true truncus arteriosus communis.

True truncus arteriosus communis Corresponding to the anlage of the four primitive bulbus ridges, a vessel usually equipped with four valves arises from both ventricles over a septal defect

Both pulmonary arteries arise as lateral branches from the ascending limb of this vessel (fig 206a) After their departure the vessel corresponds to the aorta In rare

cases there is also a ductus arteriosus but it has no functional significance. At times only one pulmonary artery develops while the other lung is supplied by dilated bronchial arteries (Humphreys) or by a persistent ductus arteriosus whose peripheral section is preserved (Soulié and coworkers, Thurnher).

These relationships indicate that the right ventricle which works against the pressure prevailing in the truncus communis, dilates and hypertrophies owing to systolic overloading. The truncus which is fed by two ventricles naturally contains mixed blood (fig. 206a). If the pulmonary branches departing from the truncus are very well developed, aeration of the blood is adequate and no noteworthy cyanosis results. On the other hand, if the two pulmonary arteries are too narrow or only

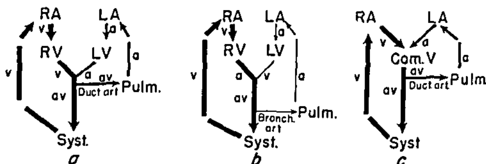


FIG. 206.—(*a*) True truncus arteriosus communis, (*b*) pseudotruncus arteriosus communis, (*c*) true truncus arteriosus communis with common ventricle.

one pulmonary artery develops, cyanosis may be present. Owing to hypoxia, the heavily burdened right ventricle undergoes considerable dilatation.

In correspondence with these different possibilities, the clinical signs vary. Cyanosis, dyspnea, enlarged cardiac dullness, precordial bulge may be present or absent. A systolic murmur and thrill over the sternum owes its development to the ventricular septal defect. The second sound at the base is loud and pure. If the valves do not close a diastolic murmur of regurgitation may be present.

Cardiac catheterization reveals the elevated pressure in the right ventricle and mixed blood in its basal part as well as in the truncus.

In typical true truncus arteriosus communis the cardiac shadow is slightly enlarged and the cardiac waist deeply excavated owing to the absence of the pulmonary artery; in the left anterior oblique position, the anterior cardiac wall shows elongation and increased rounding as evidence of right ventricular hypertrophy and dilatation. Extreme dilatation of the right ventricle and atrium, in the opinion of the author, is not a part of the picture of pure true truncus communis but is due to complications. Thus, the enlargement of the right heart (Taussig) was produced by a complicating atrial septal defect. The hilar shadows are atypical and, in particular, the left pulmonary artery which is normally clearly visible above the left main and upper lobe bronchus is not well outlined. The aortic knob is preserved and, in contrast to pseudotruncus (see below), neither abnormally large nor conspicuous.

Angiocardiography reveals immediate filling of the abnormal vessel (truncus) from the right ventricle. No trunk of the pulmonary artery can be demonstrated. Zdansky was unable to outline pulmonary arteries departing from the truncus distinctly although the atypical course of vessels entering the lungs was obvious with

opacification. The left heart shows nothing striking. Owing to refilling from the left heart, the truncus contains opaque material for an abnormally long time.

Pseudotruncus arteriosus communis. Since the division of the truncus is complete although unequal, the abnormally wide aorta (pseudotruncus) arising over the interventricular septal defect usually has three valves. There is atresia of the trunk of the pulmonary artery The lungs are supplied by collaterals, particularly bronchial arteries, which leave the arch and descending portion of the aorta and, on the left side, partly in front and partly behind the esophagus to reach the left lung. Usually no ductus arteriosus and often not even its residuum is demonstrable (fig. 206b).

There is considerable similarity between pseudotruncus and the "extreme" Fallot anomaly with atresia of the pulmonary artery (p. 299). To be sure, in the latter the lungs receive blood from a persistent ductus arteriosus, in ductus arteriosus, the truncus is usually obliterated and the lungs obtain their blood supply from ectatic bronchial arteries and other collaterals (Christeller).

The pulmonary blood supply by these collaterals is always inadequate. Consequently the blood is insufficiently aerated and cyanosis always exists

Cardiac work is not essentially different with a pseudotruncus than a true truncus except that with pseudotruncus the right ventricle must perform additional work under conditions of hypoxia, presumably this explains the conspicuous enlargement usually noted The left ventricle is also often enlarged.

Examination reveals cyanosis, considerable enlargement of the heart, precordial bulging, a loud, rough systolic murmur and thrill as the consequence of the ventricular septal defect, and a loud, pure second sound at the base of the heart

Apart from the great reduction of oxygen tension in the right ventricle and in the aorta, cardiac catheterization yields essentially the same findings as in true truncus communis

In some respects the roentgen findings differ from those of true truncus Usually the cardiac shadow is considerably enlarged, in the posteroanterior position it bulges *far to the left below the deeply excavated cardiac waist, the left lower cardiac arc* may be rounded and angular to resemble a *coeur en sabot*. However, the aortic knob is strikingly large and prominent (fig 207) since the abnormal width of the aorta is reduced to normal only after the dilated collaterals depart. Demarcation of the aortic knob is often vague owing to the departing collateral bronchial arteries (Taussig). The latter may indent the anterior and posterior walls of the barium-filled esophagus, this has diagnostic significance (Taussig, Mannheimer, Stauffer and Rigler) The roentgen picture of the heart in the left anterior oblique position is considered particularly characteristic (Taussig). In this position below the vascular shadow the heart projects almost at a right angle in a massive arc toward the anterior chest wall owing to great hypertrophy and dilatation of the right ventricle.

Since the large branches of the pulmonary artery are absent, massive, comma-shaped hilar shadows are also missing They are not demonstrable as solid shadows, rather they appear loosened and irregularly branched (Campbell and Gardner) "spider" or "crab" shape (Soulié) The absence of well defined hilar shadows indicate an abnormal vascular supply to the lungs; this may be unilateral when the opposite lung is supplied by a peripherally permeable pulmonary artery via collaterals or a persistent ductus arteriosus, this may have been the case in the patients of Cooley and Thurnher.

The close resemblance in the cardiodynamics of pseudotruncus communis and "extreme" Fallot syndrome with pulmonary atresia (p. 299) also brings a similarity of roentgen findings. In both, the enlarged cardiac shadow may show the *cœur en sabot* form although the aortic knob is usually strikingly large and conspicuous in pseudotruncus but not in the Fallot malformation.

The angiocardiogram also shows great similarity with the "extreme" Fallot. Both anomalies have in common an abnormally wide aorta which narrows to normal after the bronchial arteries depart in a pseudotruncus and after the departure of the ductus arteriosus in the Fallot anomaly. The details of the dilated bronchial arteries

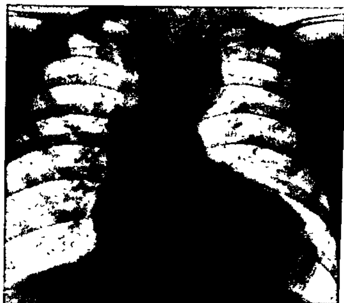


FIG 207 —Pseudotruncus arteriosus communis Boy, 9 years old, cyanotic since birth Drumstick fingers Systolic murmur over the heart Diastolic regurgitant murmur over the aorta Corrigan pulse in radial artery The cardiac shadow was considerably enlarged and widened mainly to the left and of aortic configuration with a markedly rounded left ventricular arc, conspicuous aortic knob, and a deep cardiac waist The aorta was elongated and dilated A pulsus celer was visible on the aorta Hilar shadows atypically loose and abnormally branched

and other collaterals are not distinct in the angiocardiogram but sometimes a ductus arteriosus may be seen in the left anterior oblique position. The abnormal vascular supply of the lungs becomes more obvious with contrast filling.

Surgical intervention does not come into consideration in a truncus arteriosus communis. In pseudotruncus the Blalock-Taussig operation seems justified in principle after careful evaluation of the indications. The situation is less favorable only in so far as the collaterals with which the anastomosis is created are usually narrow and the pressure is the same as that prevailing in the subclavian artery.

Truncus or pseudotruncus arteriosus communis with absence of the ventricular septum Truncus as well as pseudotruncus may be associated with absence of the ventricular septum. The atrial septum may also be missing. The large solitary ventricle then propels its content into a large vessel which supplies the coronary, systemic and

pulmonary circulations If a ductus arteriosus persists, the blood supply for the lungs is ensured (fig. 206c). If, on the contrary, the ductus is absent, the lungs can be fed only by collaterals (upper and posterior bronchial arteries, mediastinal and esophageal arteries) (Christeller), then aeration of the blood is insufficient

Examination usually reveals deep cyanosis and severe dyspnea The capacity of the individual for exertion is limited to a variable degree depending upon the development of collaterals The heart is normal in size or slightly enlarged No murmurs are heard and the second sound at the base of the heart is pure

Cardiac catheterization reveals a large ventricular space (Bing) In it, in the great vessels departing from it, and in the femoral arteries, oxygen tension is identical

The author has no observations established by necropsy or by clinical findings and consequently must rely upon the roentgen findings mentioned by Taussig The cardiac shadow is normal in size or a little enlarged The shape is not characteristic Rotation of the heart is common and may suggest dextrocardia In the early years of life the heart may look like one of the Fallot malformations although usually the aortic knob is distinctly enlarged and prominent In the absence of a ventricular septum the heart should not bulge markedly toward the anterior chest wall in the left anterior oblique position as happens in pseudotruncus with an intact interventricular septum If the heart descends with the diaphragm as the child grows older, its size and shape become progressively less striking. Nevertheless one may note a considerably enlarged aorta which courses over the left or right bronchus

Angiocardiography reveals a large solitary ventricle as well as the course and considerable width of the single departing vessel.

The Blalock-Taussig operation, in case it is technically feasible, may help greatly

9 Anomalies from Improper Torsion of the Truncus-Bulbus Division

For the correct implantation of the two chief arteries in appropriate ventricles and for the normal interrelationships of these two vessels, proper torsion of the bulbus-truncus septum and of the ventricular septum is fundamental (Pernkopf and Wirtinger)

If no torsion occurs or it is insufficient in the bulbus-truncus area an abnormal junction of the vessels with the ventricles results. The extreme outcome of this anomalous union is complete transposition of the great arteries (v Rokitansky), the venous tricuspid ventricle joins the aorta and the arterial bicuspid ventricle joins the pulmonary artery.

If the bulbus-truncus septum undergoes some, but inadequate, torsion, dextroposition of the aorta or sinistroposition of the pulmonary artery occurs, in this situation the affected vessel arises from both ventricles over a rather high septal defect Dextroposition of the aorta ("riding aorta") is definitely more common than sinistroposition of the pulmonary artery ("riding pulmonary artery"). No cases with both vessels astride a single septal defect seem to have been reported in roentgenologic literature. On the other hand, aortic dextroposition may be associated with transposition of the pulmonary artery or a sinistroposition of the pulmonary artery with aortic dextroposition.

Through unequal division of the truncus or from abnormal hemodynamic conditions resulting from shunts in the heart and between the vessels, differences in the diameters of both great vessels may occur and result in more or less extreme stenosis and even atresia of one.

The important forms of anomalous torsion of the bulbus-truncus section which are conducive to abnormal anatomic structure and for abnormal cardiodynamics are discussed below

These forms are associated with innumerable transitions They merely indicate more or less characteristic types which differ somewhat in respect to their variable consequences for the heart and the pulmonary and systemic circulations as well as their differing susceptibility to surgical management Since the anatomic basis of these different forms flow into each other, the roentgen findings including angiocardiography also show transitions This also prevails for the functional consequences discovered by the clinician and finally for the indications for surgical intervention Only most careful reflection in which the roentgen findings play an important part reveal these indications.

Complete transposition of the great vessels. This anomaly affects the male sex by preference It has the following results in extrauterine life· blood from the right ventricle passes through the aorta into the systemic circulation and returns through both venae cavae to the right atrium and ventricle, blood from the left ventricle is pumped through the pulmonary artery to the lung circulation and hence returns to the left atrium via the pulmonary veins and reaches the left ventricle This situation would be incompatible with life owing to complete independence of the two circulations, life is possible only when communications between the two circulations permit exchange of blood As communications, the ductus arteriosus, the foramen ovale, an atrial or ventricular septal defect or some combination of them comes under consideration.

The presence and the width of these communications determines the extent of exchange between the two circuits, the degree as well as the distribution of blood. The combination and width of these junctions is also decisive for pressure relations in the single divisions of the heart and therefore for their dilatation and hypertrophy. Moreover, dilatation is especially determined by the severity of hypoxemia and the duration of life. At birth the heart is not enlarged since the oxygen supply of the organism in fetal life is not disturbed by a complete transposition The highest average duration of life (10 years and more) is found when a ventricular septal defect and a patent foramen ovale coexist, life is shorter when a ventricular septal defect constitutes the sole communication After birth, both sides of the heart dilate, and hypertrophy after the institution of respiration and unfolding of the lungs is associated with a reduction of pressure in the pulmonary artery and in the left half of the heart Consequently blood passes from the right half of the heart into the left through an atrial or ventricular septal defect or, in the presence of a persistent ductus arteriosus, from the aorta into the pulmonary artery In either instance filling increases and pressure rises in the left side of the heart and there is progressive emptying and reduced pressure in the right half of the heart and the systemic circulation. Ultimately a point is reached at which the pressure in the left heart and pulmonary circulation exceeds that in the right heart and aorta, then, the shunt must

reverse. Owing to this reversal the right heart and systemic circulation experience greater filling which lasts until pressure has become greater than in the left heart and the pulmonary artery, then the direction of flow in the shunt again reverses The steady change results in periodic alteration of atrial volume (Taussig)

If more than one communication connects the two circulations, if there is a septal defect and a persistent ductus arteriosus for example, periodic reversal of the shunt may be absent, one pathway *may lead to a constant right to left shunt and* the other to a steady left to right (Cooley and Sloan). Then periodic volumetric changes of the atria do not occur.

At all events, both atria and both ventricles dilate and hypertrophy through constant or periodic variations in heightened resistance and augmented filling.

As noted above, the extent of dilatation is profoundly influenced by the degree of hypoxia. Under otherwise equal conditions, with increasing depth of the cyanosis there is greater development of the *universal dilatation of incompetence* (p *141*). Finally signs of manifest failure of one or both ventricles appear with evidence of retrograde congestion in the associated atrium, in the lungs, or in the systemic circulation

Corresponding to the variable connections between the atria, ventricles, and great vessels, the degree and distribution of cyanosis differs in complete transposition In the first few days or weeks

Moreover, the murmur and palpable thrill vary according to the presence and the size of the *different communications between the two circulations, they are not characteristic*

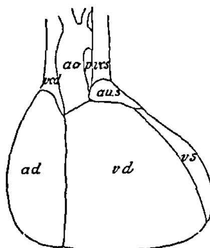
Cardiac catheterization reveals elevated pressure in the right atrium and ventricle as well as the possibility of entering the aorta If an atrial or ventricular septal defect exists there is the possibility of entering the left half of the heart or the pulmonary artery respectively Moreover a persistent ductus arteriosus has been demonstrated in this way

The oxygen tension in both halves of the heart may differ and vary according to the direction of flow in the shunt

The roentgen findings of complete transposition of the great vessels show astonishing variability To some extent, this depends upon differing grades of dilatation and hypertrophy of the two halves of the heart, on the other hand differing intracardiac communications between the two circulations as well as the varying *reciprocal relations in the position of the two great arteries exert a decisive influence* The moderate to considerable enlargement of the heart is combined with an elongation and increased rounding of both cardiac borders. In the left anterior oblique position there is marked protrusion toward the anterior chest wall as well as into the vertebral shadow, this may be explained by the dilatation and hypertrophy of both ventricles. The elongation of the markedly rounded right cardiac border may be ascribed to right atrial enlargement Often the posterior cardiac wall bulges distinctly into the posterior mediastinum, locally indenting the *barium-filled esophagus*, this can be explained by left atrial enlargement. Diastolic overload is responsible for the enlargement of the atria Sometimes, by fluoroscopy one can observe periodic changes in atrial volume which result from the previously described periodic variations of pressure in the right and left halves of the heart (Taussig).



a



b

The left border of the vascular band is formed by the persistent left superior vena cava (vcs). The bronchial arteries are markedly dilated. The ductus Botalli is patent. (From Assmann, *Klinische Rontgendiagnostik*)

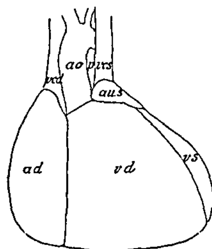
With large atrial septal defects and with the combination of several shunts, these fluctuations of atrial volume are missed.

Usually the supravascular shadow is short. Its varying width and shape account for much of the diversity of roentgen findings. In some cases with p-a position the short vascular shadow looks like a very small appendage of the cardiac shadow (fig. 208a and b). In them the base of the aorta arises in front of the pulmonary artery and the ascending aorta rises almost median (Fanconi, Taussig), bending dorsad at the arch to pass over the left bronchus (fig. 209b). With rotation to the left anterior oblique position the aorta is seen to arise ventrad from the heart, thereby the vascular band increases in width since the aorta is projected outward by the pulmonary artery which is located behind Castellanos and coworkers call this type II transposition. In other cases the vascular band is about normal in width in the p-a view but becomes smaller in the right anterior oblique position. This happens when the aorta, arising from the right ventricle does not start in front but to the right of the pulmonary artery (fig 209a). This is called type I transposition (Castellanos et al) Finally, the transposed aorta may arise to the left of the pulmonary artery from a conical outflow path of the right ventricle (figs 191 and 209c) (Rossi and Prader, Campbell and Hill, Donzelot and coworkers, Zdansky, Thurnher and Weissel, and others) The two authors mentioned last regard this course of the arteries as reflecting the inversion of the bulbus-truncus section. In these cases the ascending aorta curves convexly to the left forming the left border of the vascular band which is often unusually broad The cardiac waist is filled by the conus of the right ventricle and the ascending aorta in such a way that one might assume a dilatation of the pulmonary artery Then, the ascending aorta ascends median to pass over the left (Castellanos type III) or right bronchus (Castellanos type IV) In the p-a position the right border of the vascular band proceeds in a straight line or slightly *concave and scarcely projects over the spinal shadow since the ascending aorta is* missed on the right side and ascends in the left mediastinum (Thurnher and Weissel) (fig 191)

Angiocardiography clarifies the reciprocal relations of the two vessels. The *aorta, arising ventral to the heart, is immediately and intensely filled from the right* ventricle Definition of the pulmonary artery depends upon very variable intensity of its filling, its position and width. Often angiocardiograms in two planes are required for its demonstration When the pulmonary artery is narrow or, on the whole, cannot be defined, differentiation from an "extreme" Fallor or a *Taussig-Bing* anomaly is difficult or impossible, then, the angiocardiogram does not prove with certainty whether the aorta is transposed or merely dextroposed and rides over a ventricular septal defect The same holds for the pulmonary artery. The demonstration of intracardiac shunts is often difficult or impossible in the angiocardiogram. Even large atrial or ventricular septal defects can escape detection or can merely be assumed. Their demonstrability depends not only on their diameter, which is frequently small, but also on the continuous or intermittent direction of the stream through the shunt which may be unfavorable for contrast filling. Favorite described a rare example of so-called "mixed arteriovenous transposition" (Spitzer). The aorta arose from a very small right ventricle. The pulmonary artery emerged from a very large left ventricle which housed both atrioventricular valves and communi-



a



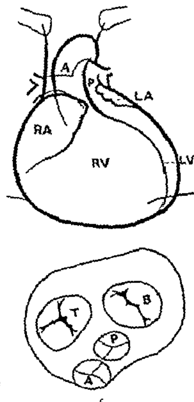
b

Fig 208 —Crossed transposition of the arteries (with persistence of left superior vena cava and persistent ductus Botalli (necropsy) (*a*) Child of seven weeks with marked cyanosis, systolic murmur over the apex and over pulmonary and slight accentuation of the second pulmonic sound

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to the right. In the left anterior oblique position the right position of the aorta would be demonstrable.

All these forms of transposition are *frequently* associated with *right position of* the aortic arch or represent an additional feature of some other profound disturbance of cardiac architecture Sometimes the transposition of the vessels provides a perfect or partial correction of anomalous circulation as in certain partial *inversions* of the heart (p 311) or tricuspid atresia (p 275).



(*Fig 209 cont from p 296*)

c

the pulmonary artery In the anterior view the *aorta* and pulmonary artery are projected into each other In this way the vascular band becomes remarkably narrow It is wider in the left anterior oblique position (*c*) The aorta arises at the *left* and in front of the pulmonary artery and bulges to the left In the anterior view the vascular band is abnormally broad In the left anterior oblique position it is smaller

Transposition of the aorta with sinistroposition of the pulmonary artery (Taussig-Bing syndrome) In this rare anomaly the aorta arises from the right ventricle The pulmonary artery is not completely transposed but is merely displaced sufficiently to arise from two ventricles over a defect located high on the interventricular septum The pulmonary artery contains blood from both ventricles, usually it is dilated but sometimes it is hypoplastic The right ventricle, which ejects most of its content into the aorta and a smaller portion into the riding pulmonary artery, dilates and hypertrophies from systolic as well as diastolic overloading The blood, arterialized in the lungs, returns to the left ventricle via the left atrium, the former advances its con

cated with the right small aortic ventricle through a septal defect. Since the left ventricle received blood from both atria, it was dilated and hypertrophied, moreover, its pulmonary artery was greatly dilated. The patient attained the remarkable age of 18 years and died suddenly from rupture of the pulmonary artery. The broadly permeable ductus arteriosus and large ventricular septal defect united the two circulations. Roentgen examination was undertaken only after the pulmonary artery had ruptured. It disclosed a cardiac shadow widened considerably to the right

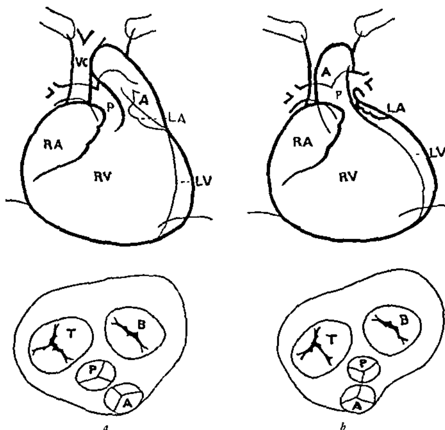


Fig 209 —Different forms of transposition of the great vessels (*a*) The aorta arises at the right and in front of the pulmonary artery and ascends cephalad at the right and in front of the pulmonary artery The vascular band is normal in width in the posteroanterior position It is smaller in the right anterior oblique position (*b*) The aorta arises almost purely ventrad from (*continued on page 297*)

and left with a conspicuous pulmonary arc Naturally the picture was drastically influenced by the intrapericardial hemorrhage and exact roentgen analysis was impossible. It may be assumed that this anomaly leads to an enlargement and globular shape of the cardiac shadow with prominence of the pulmonary arc. The vessel markings in the lungs should be accentuated and should show marked systolic expansile pulsations. The diameter of the aorta would be small, in striking contrast—of diagnostic significance—to the demonstrable size of the left ventricular cavity. The right cardiac border should show powerful pulsations transmitted from the left ventricle and might also be subdivided since the pulmonary ventricle would extend

The oft-employed expression "tetralogy of Fallot" may be traced to the fact that the anomaly has been considered *the sum of four features* riding aorta, ventricular septal defect, pulmonary stenosis and hypertrophy of the right ventricle. In *reality the overriding aorta and the interventricular septal defect are a genetic unit,* moreover right ventricular hypertrophy merely represents the inevitable outcome of greater effort when this ventricle labors against increased resistance from the pulmonary stenosis and by virtue of its added work against aortic pressure In the rare cases of so-called "extreme Fallot" (Taussig) with a functionally or anatomically closed pulmonary artery, a ductus arteriosus persists to supply the pulmonary circulation, moreover, dilated bronchial arteries may assist in supplying blood to the pulmonary circulation.

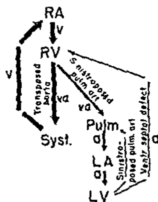


Fig 210

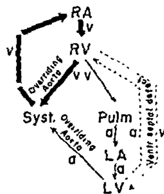


Fig 211

Fig 210 — Transposition of the aorta and sinistroposition of the stenotic pulmonary artery *(Taussig-Bing's anomaly)*

Fig. 211 — Dextroposition of the aorta with stenosis of the pulmonary artery (Fallot's anomaly)

Inasmuch as there is a pulmonary stenosis, the aorta overriding the interventricular septal defect receives an abnormally large quantity of blood from both ventricles The mixed blood flows from the aorta into the systemic circulation and returns into the right atrium The small quantity of venous blood entering the pulmonary artery *becomes oxygenated in the lungs and returns through the left atrium into the left* ventricle. From the left ventricle it is pumped into the overriding aorta and partly into the right ventricle as well as by way of the interventricular septal defect if pressure in the left ventricle exceeds that in the right (fig. 211) Since the quantity of oxygenated blood which the aorta receives from the right ventricle is relatively small, owing to the pulmonary stenosis, hypoxemia develops. The cyanosis is deeper with greater displacement of the aorta over the right ventricle and with *greater stenosis of the pulmonary artery.* Owing to work against aortic pressure and through elevated resistance in the pulmonary artery, the right ventricle dilates and hypertrophies but this remains modest since the patients are poorly equipped for exertion and necessarily spare their hearts.

Examination reveals deep cyanosis, polycythemia, and clubbed fingers as well as toes. Cyanosis often appears soon after birth as the ductus arteriosus closes. In most cases, however, it is distinct

tents, in part, again into the pulmonary artery and in part through the ventricular septal defect into the right ventricle (fig. 210). The relatively small amount of arterialized blood entering the left ventricle is inadequate for the oxygen supply of the organism

Cyanosis is intense and a loud, rough systolic murmur is heard over the sternum

Cardiac catheterization reveals high pressure in the right ventricle through which the aorta may be immediately entered While the inflow tract of the right ventricle shows a venous O_2 tension, the outflow tract and the aorta contain mixed blood At times the pulmonary artery also can be entered from the right ventricle and contains mixed blood

The moderately enlarged heart is usually characterized by conspicuous dilatation of the pulmonary artery in the cardiac waist The enlargement primarily involves the right atrium and the right ventricle from which the aorta and part of the pulmonary artery arise In the left anterior oblique position bulging of the anterior cardiac wall and the ventral origin of the aorta are distinct. Usually the hilar shadows are enlarged by dilatation of branches of the pulmonary artery and display increased pulsations Great similarity to an Eisenmenger complex (p. 307) would result if the aorta were not dextroposed This relation, decisive in differential diagnosis, is easiest to detect in angiocardiograms The opaque material immediately and intensely fills the aorta while the pulmonary artery is faintly opacified, the opposite situation prevails in Eisenmenger's anomaly since the pulmonary artery is filled fully while the aorta is faint

In the rare case with a hypoplastic sinistroposed pulmonary artery (Braun and coworkers), the pulmonary arc is not prominent, the hilar shadows are small and the vascular markings sparse

Only in these rare cases does the Blalock-Taussig operation come under consideration, it should be noted, however, that an artificial ductus arteriosus might overload the sorely taxed and dilated right ventricle.

Dextroposition of the aorta with normal position of the stenotic or atretic pulmonary artery (Fallot's anomaly) The dextroposition by virtue of which the aorta rides a defect high on the ventricular septum, is usually associated with pulmonary stenosis or, more rarely, pulmonary atresia There are all transitions between cases in which the aorta arises mainly from the left ventricle to those in which it arises largely from the right

Moreover, pulmonary artery stenosis may vary between a slight grade and complete atresia. Finally, the location of the stenosis varies. In most cases it involves the infundibulum which resembles a narrow channel or a funnel, less commonly, the stenosis is located at the valves which may be malformed or deficient in number; occasionally, infundibulum as well as the valves are affected Moreover, if the pulmonary artery is atretic, the stenosis can involve the infundibulum, the ostium pulmonale, or the pulmonary artery (Burke, Kirklin, and Edwards). The entire group are mere variants of the Fallot anomaly All cases in the group have the following features in common

1. A defect high on the interventricular septum with overriding (dextroposed) aorta
2. Stenosis or atresia of the pulmonary artery.

of the left cardiac are near the base and particularly its apical part which falls sharply to the diaphragm now belong to the right ventricle (fig. 197*b*). The left ventricle now forms the contour to an indefinite extent for a short section in the middle of the left cardiac border. The differentiation from hypertrophy of the left heart is made even more difficult since in the left anterior oblique position one gains the impression that the left ventricle is also enlarged. In this position the left cardiac border tends to bulge into the vertebral shadow as with left ventricular enlargement while the anterior cardiac border descends rather vertically to the diaphragm. Only with great dilatation of the hypertrophied right ventricle, a more common event in extreme dextroposition (Taussig) and in severe stenosis of the pulmonary artery, does a rounded cardiac shadow bulge toward the thoracic wall



FIG 213 — Tetralogy of Fallot with right aortic arch Male, 24 years old, with deep cyanosis and clubbed fingers Red blood cell count 7.2 millions. Sahli, 138, loud systolic murmur with maximum intensity over Erb's point Electrocardiogram dextrocardiogram The left cardiac border showed the typical rounded kinking of cœur en sabot The aortic arch passes over the right bronchus The descending aorta forms the right border of the vascular band

Right ventricular hypertrophy and dilatation in Fallot's anomaly produces an entirely different cardiac shape than in a mitral lesion, cor pulmonale, pure pulmonary stenosis or atrial septal defect. In these cases, elongation and expansion of the outflow tract of the right ventricle and dilatation of the pulmonary artery fill the cardiac waist and consequently create a mitral configuration, in the Fallot anomaly the heart is transverse and the cardiac waist preserved (fig. 197a and b) This difference is not solely dependent on stenosis of the pulmonary artery, it is intimately connected with the varying anatomic structure of the right ventricle and the particular hemodynamic conditions of this anomaly The massively developed crista supraventricularis projects as a strong bridge between the stenotic pulmonary artery and the aorta riding the septal defect, it directs the blood stream of the right ventricle toward the aorta (A. Spitzer). This causes a further curvature of the outflow tract of the right ventricle in favor of the abnormal outflow tract located between the apex of the right ventricle and the ventricular septal defect. It is important for the hemodynamic transformation of the heart that the axis of this new outflow tract does not rest perpendicularly on the diaphragm (as with a normal right ventricle) but

only when the child begins to walk. Limitation of physical activity is rather extreme. The child prefers the squatting position, and physical development is retarded. Usually the left anterior chest wall bulges, although cardiac dullness is not enlarged. There is a loud, rough, systolic murmur over the sternum and to its left. A thrill may be felt at the base of the heart. The second pulmonic sound is soft, the aortic second sound is often distinctly accentuated and sometimes palpable.

The prognosis depends primarily upon the degree of pulmonary stenosis. When cyanosis appears only after the ductus arteriosus closes or only in the second to third month of life, prognosis may be more favorable than in patients severely cyanotic soon after birth. The outlook has been definitely improved by the possibility of the Blalock-Taussig operation.

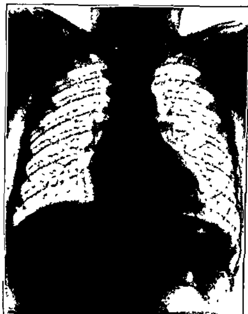


FIG. 212.—Fallot's anomaly (necropsy). This 15 year old individual had cyanosis and clubbed fingers. The red blood cell count was 9.3 millions, hemoglobin 139 per cent, loud, drawn-out systolic murmur over the pulmonic. The cardiac waist was well maintained. Pulmonary arc flat. In its lower third, the left cardiac border *descends below a round angulation mesially to the* diaphragm (coeur en sabot). Vascular markings in the lungs delicate. (From Assmann, *Klinische Rontgendiagnostik*.)

Cardiac catheterization in this anomaly reveals elevated right ventricular pressure. Usually the catheter first enters the aorta which contains mixed blood. Occasionally, however, the narrow pulmonary artery and the left cardiac cavity are also entered through the ventricular septal defect.

Roentgenologically, no mitral configuration results from the resistance dilatation and hypertrophy of the right ventricle and the cardiac waist remains excavated and the left cardiac border is rounded and angular (W. Raab), below it, the cardiac border descends obliquely and somewhat median to the diaphragm (figs. 212 and 213). [illegible]
This f[illegible]
and th[illegible]
sabot from resemblance to a wooden shoe. The shape develops in the following manner. the hypertrophied right ventricle contributes *widely to the left cardiac* border since, in developing to the left, it pushes the left ventricle dorsad. The part

can widen the cardiac shadow to the left; thereby the cardiac apex, formed by the right ventricle, is displaced to the left and the left ventricle is thrust backward.

In a typical Fallot anomaly the hilar shadows are small and the intrapulmonic vascular markings are sparse corresponding to the defective perfusion of the lungs. If the pulmonary circulation receives blood from ectatic bronchial and other arteries of the mediastinum, owing to extreme pulmonary stenosis or atresia, no typical, comma-shaped hilar shadows are seen (fig. 214a and b) The hilar shadows look loose, branch irregularly, and are surrounded by round spots corresponding to cross sections of abnormal vessels Since the bronchial arteries may depart from the aortic arch and the initial part of the descending aorta, the abnormal vessels may also appear above the roots of the lungs to pass into the mediastinum (Campbell and



FIG 215 —Angiocardiogram of a typical Fallot anomaly

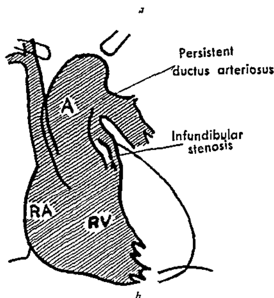
Gardner) In an "extreme" Fallot, Zdansky saw an abnormally large, comma-shaped left hilar shadow which could be attributed to the left pulmonary artery, dilated by a persistent ductus arteriosus (fig 215)

At times a poststenotic dilatation of the pulmonary trunk causes the pulmonary arc to project into the cardiac waist (Baumgartner and Abbott) The resultant resemblance to the Eisenmenger complex is great; however the hilar shadows are not enlarged and do not pulsate.

In some cases left ventricular dilatation seems to predominate Originally this was described in a patient (Weinberg and Wiesner) with an associated large atrial septal defect These cases have been called the "pentalogy of Fallot." Obviously, the addition of an atrial septal defect permits a large amount of blood under higher pressure in the right atrium to pass into the left and produce a diastolic overload of the left heart with dilatation and hypertrophy. These hearts can show an aortic configuration since the hypertrophied and dilated left ventricle determines the shape



a



b

Fig 214.—Extreme Fallot with persistent ductus arteriosus Boy, 11 years old, who had been cyanotic since birth Clubbed fingers Severe dyspnea Loud, rough, systolic murmur over the heart with point of maximum intensity over Erb's point No definite diastolic murmur.

proceeds obliquely from the apex of the ventricle to the right, posterior and above toward the common aortic ostium (fig 197a and b) These differing courses of the outflow tracts transform the heart in different ways because a mitral configuration occurs only when the outflow tract descends almost perpendicularly to the diaphragm (p 143), on the contrary, an outflow tract running obliquely from the left, anterior and below, to the right, posterior and above, by elongation and expansion

cerning the presence and the distribution of the large branches of the aortic arch, this has importance in the anastomoses

In "extreme" Fallot syndrome with atresia or severe stenosis of the pulmonary artery, the very marked *dextroposition*, unusual width, and intense opacification of

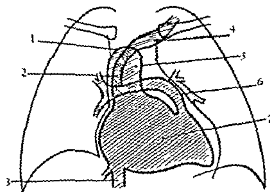


FIG 217 – Angiocardiogram of a Fallot's tetralogy in anterior view (After Sussman and Grishman)

1 Aortic arch
2 Sup vena cava
3 Inf vena cava
4 Left innominate vein
5 Ascending aorta
6 Pulmonary artery
7 Right heart

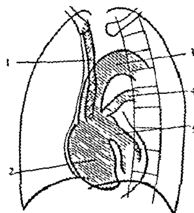


FIG 218 —Angiocardiogram of Fallot's tetralogy in left anterior oblique view (After Grishman, Steinberg, and Sussman)

1 V cav sup
2 Right ventricle
3 Aortic arch
4 Pulmonary artery
5 Passage of opacified blood from the right ventricle into the left

the ascending aorta are striking. Usually the pulmonary artery cannot be outlined The hilar shadows are particularly small, loose, and atypical in shape when ectatic bronchial and mediastinal arteries supply the lungs (Soulié, Campbell and Gardner). If the ductus arteriosus persists, the hilar shadows, particularly the left, may even be enlarged

of the left cardiac border, the wooden shoe shape of the heart is more or less effaced in this way.

An essential mark of the Fallot anomaly is dextroposition of the aorta. This may be detected even in posteroanterior views when the aorta forms the entire right border of the vascular band. It is even more distinct in the left anterior oblique position since the pulmonary stenosis makes the aortic window abnormally clear except rarely when there is a poststenotic dilatation of the pulmonary artery.

About one fourth of the patients have an arcus aortae dexter (p. 418). Its recognition is of interest to surgeons and therefore demands attention.

The angiocardiogram reveals important information concerning communications between the ventricles and arteries. Thus, immediately after the contrast substance fills the right atrium and ventricle, the aorta riding the ventricular defect fills

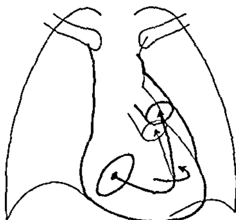


FIG. 216.—Dilatation and hypertrophy of the outflow path of the right ventricle from increased resistance. Through elevation of the dilated conus and pulmonary artery there is mitralization of the heart without enlargement of the transverse diameter. The rotation of the heart to the left (curved arrow) favors filling of the cardiac waist.

simultaneously with the pulmonary artery (figs. 216 and 217). In the left anterior oblique position, a stream of blood containing the contrast agent is often seen passing from the right into the left ventricle (fig. 218). Angiocardiograms reveal, in general, that the aorta is widened in proportion to the degree of pulmonary artery stenosis, moreover the filling of the aorta is more intense and the pulmonary artery fainter as dextroposition of the aorta increases. Often the localization and extent of pulmonary stenosis is not detectable. Experience has shown that in the Fallot anomaly the stenosis is usually located in the region of the infundibulum, nevertheless this localization can be established on the angiocardiogram only when the outflow tract of the right ventricle is distinctly narrowed not only in the systolic but also in the diastolic phase of the heart cycle. Corresponding to the pulmonary stenosis, the arteries in the hiluses and lungs are narrow. Their width in the angiocardiogram has surgical interest because an anastomosis of a subclavian or innominate artery with a pulmonary artery below a certain caliber creates technical difficulties. Owing to improper perfusion of the pulmonary circulation, the left heart cavities often show only faint opacification. Finally, the angiocardiogram provides information con-

through the septal defect into the overriding aorta, the same holds when the pulmonary circulation is fairly copious owing to a persistent ductus arteriosus. If the ventricular septal defect is small and the aorta arises almost exclusively from the right ventricle, this ventricle dilates and hypertrophies as the left anterior oblique position reveals by the marked bulging toward the anterior chest wall. In this position the displacement of the aorta to the right and forward as well as the striking clarity of the aortic window is apparent.

In the angiocardiogram the riding aorta becomes immediately opaque from the right ventricle. The left heart and the pulmonary artery are poorly defined and the

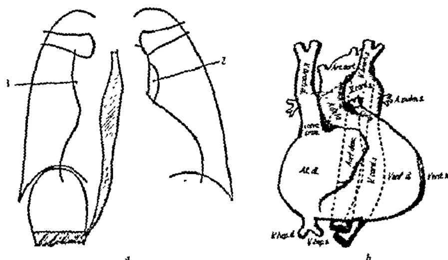


FIG 220 —Situs inversus partialis abdominalis with dextroposition of the aorta transposition of the stenotic pulmonary artery, persistent left cardinal vein and aplasia of the inferior vena cava (necropsy) See text for description (a) Anterior view, (b) findings at necropsy

1 T brachiocephalica dextra 2 V cardinalis sinistra

latter is, like the pulmonary vessels, belatedly and faintly filled The angiocardiogram shows the ventral position of the aorta very distinctly in the left anterior position

The prognosis after surgical treatment is not favorable in spite of a postoperative improvement of the oxygenation of the blood, because the marked dextroposition of the aorta hinders the entrance of oxygenated blood from the left ventricle into the aorta

Dextroposed aorta with normal position of a pulmonary artery, normal in width or dilated (Eisenmenger's complex) In Eisenmenger's complex the aorta is dextroposed over a high ventricular septal defect, the origin of the pulmonary artery is normal and its width also normal or increased. Often the aortic valves are deformed and sometimes they fail to close. The pulmonary valves may become relatively incompetent owing to dilatation of the base of the pulmonary artery In accordance with the amount of aortic dextroposition, some venous blood may pass from the right ventricle into the aorta, but the largest part is pumped through the pulmonary artery into the lungs The oxygenated blood returns from the lungs into the left heart and is

For the surgical treatment of Fallot's anomaly, Blalock undertook the creation of an artificial ductus arteriosus upon the suggestion of Taussig, an end-to-side anastomosis of the subclavian or innominate artery with the pulmonary artery was performed. Cyanosis, polycythemia, and clubbing of the fingers may recede decidedly and the physical capacity and development of the individual may approach normal Naturally, the operation is not equally appropriate for all patients. With extreme dextroposition of the aorta, pulmonary circulation, though improved by the operation, usually remains inadequate for properly supplying oxygen to the organism, it is difficult for aerated blood in the left ventricle to enter an aorta which departs largely from the right ventricle. In other cases the pulmonary artery is extremely narrow and creates difficulties in performing an anastomosis.

Dextroposition of the aorta with transposition of the stenotic or atretic pulmonary artery In this rare anomaly the dextroposed aorta arises over an interventricular septal defect largely from the right ventricle and only for a minor part from the left ventricle while the transposed pulmonary artery departs exclusively from the left ventricle.

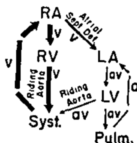


Fig 219 —Dextroposition of aorta with transposition of stenotic pulmonary artery

The communication between the two circulations is made possible by an atrial septal defect and the highly situated interventricular septal defect over which the aorta rides, sometimes the ductus arteriosus is patent.

The left atrium is filled by way of the pulmonary veins and by the atrial septal defect. The mixed blood flows then into the left ventricle which ejects its content partly into the narrow transposed pulmonary artery and partly into the overriding aorta. The blood which enters the pulmonary circulation returns oxygenated into the left atrium and ventricle. It is in this way that the aorta receives a small quantity of oxygenated blood Since the aorta arises chiefly from the right ventricle, the greatest part of the venous blood enters the aorta (fig. 219). From this results severe hypoxemia, and death usually occurs in the early days or weeks of life. Those with a large ventricular septal defect or a persistent ductus arteriosus live longer.

The systolic murmur and thrill is associated with considerable cyanosis and dyspnea

The cardiac catheter passes directly from the right ventricle into the aorta. Intra-aortic O_2 tension is only a little higher than in the right ventricle The left atrium may be entered through the atrial septal defect and contains mixed blood In rare cases the pulmonary artery may be entered through the ventricular septal defect

The resemblance of the x-ray picture to a Fallot anomaly is close (fig. 220) The cardiac waist is preserved. The heart is almost normal in size or a little enlarged when relatively large amounts of oxygenated blood pass from the left ventricle

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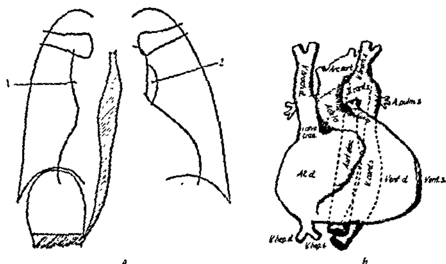


FIG 220—Situs inversus partialis abdominalis with dextroposition of the aorta, transposition of the stenotic pulmonary artery, persistent left cardinal vein and aplasia of the inferior vena cava (necropsy). See text for description (a) Anterior view, (b) findings at necropsy

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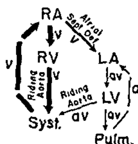


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dilated pulmonary artery is seen (fig 222) The initial filling of the aorta is decidedly less than that of the pulmonary artery, this is significant in differentiation from a Taussig-Bing syndrome which presents the opposite situation (p 298). After the opaque material passes through the pulmonary circulation there is intense filling of the left cardiac cavities and secondary opacification of the aorta from the left ventricle. Thus, the aorta is refilled.

Operation does not come under consideration.

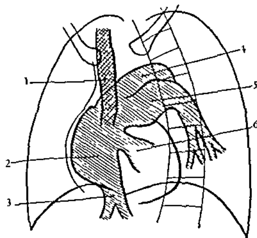


FIG 222 —Angiocardiogram of an Eisenmenger complex in the left anterior oblique position (After Rossi and Prader)

1. V cava sup
2. Right heart
3. V cava inf
4. Aorta
5. Pulmonary artery
6. Passage of opacified blood from the right into the left ventricle

10 *The Heart in Situs Viscerum Inversus*

In situs viscerum inversus there is the mirror-image anlage of organs and structures which normally develop asymmetrically. A distinction is made between situs viscerum inversus totalis and partial inversion, in the former, all organs of the chest and abdomen are laid down inversely In situs inversus partialis thoracalis the thoracic organs and in situs viscerum inversus partialis abdominalis the abdominal organs are laid down inversely In a situs viscerum inversus partialis not all organs and structures of the chest or the abdomen are always inverse, some of them may be found in a normal location Thus, single parts of the heart may be inverse while others are in their customary location

Situs viscerum inversus totalis The simplest relationships exist in situs viscerum inversus totalis. In this form all organs of the thorax and abdomen are laid down in a mirror image so that the anomaly is devoid of significance from the standpoint of circulatory physiology (fig. 223b) Fluoroscopy reveals the true mirror image of the normal cardiac shadow, the gastric air bubble is located under the lower right diaphragm, the liver under the higher left diaphragm. *Occasionally a left sided*

pushed from the left ventricle into the riding aorta (fig. 221). There is little or no cyanosis.

A loud, rough, systolic murmur and thrill is found over the base of the heart as the result of the ventricular septal defect. Sometimes a soft, blowing diastolic murmur originates from a coexisting aortic or pulmonary valvular regurgitation The second pulmonic sound tends to be accentuated Usually cyanosis is missed in childhood but gradually appears later in varying degrees The influx of venous blood from the right ventricle into the aorta does not account for it, rather defective gas exchange in the pulmonary circulation is partly responsible (Taussig) When dilatation of the pulmonary artery is marked, deep cyanosis tends to occur more frequently and clubbing of the fingers may also appear Occasionally hemoptysis is noted

Cardiac catheterization reveals elevated pressure in the right ventricle and the pulmonary artery, this speaks strongly against a pulmonary stenosis The pulmonary artery and the aorta can be entered from the right ventricle Intra-aortic oxygen tension is reduced more or less according to the degree of dextroposition.

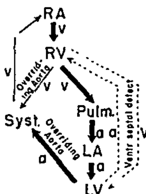


FIG 221 —Dextroposition of aorta with normal arising, pulmonary artery of normal width or dilated (Eisenmenger's anomaly)

The heart is moderately enlarged to left and right as the result of hypertrophy and dilatation of both ventricles, the cardiac borders show lively pulsations. A mitral configuration may result from the conspicuous bulge of the pulmonary arc owing to dilatation of the pulmonary artery The hilar shadows are somewhat enlarged and the pulmonary vascular markings accentuated They may display increased, systolic-expansile pulsations particularly when pulmonary regurgitation is present. If the aortic valves are incompetent, the aortic configuration is more prominent and usually an aortic pulsus celer is distinct. The roentgen picture may closely resemble that of an atrial septal defect, a defect located high on the ventricular septum, a mitral lesion with or without relative insufficiency of the pulmonary valve, a Fallot anomaly with poststenotic dilatation of the pulmonary artery or a Taussig-Bing malformation The absence of left atrial enlargement speaks against a mitral or a mitral-aortic lesion; the enlargement as well as systolic expansile pulsations of the hilar shadows speaks against pulmonary stenosis of a Fallot anomaly. Often differentiation from an atrial or ventricular septal defect is impossible. Not rarely angiocardiography and cardiac catheterization must be employed for differential diagnosis

The angiocardiogram shows simultaneous opacification of the pulmonary artery and aorta from the right ventricle. Dextroposition of the aorta and the normal or

dilated pulmonary artery is seen (fig. 222) The initial filling of the aorta is decidedly less than that of the pulmonary artery, this is significant in differentiation from a Taussig-Bing syndrome which presents the opposite situation (p. 298). After the opaque material passes through the pulmonary circulation there is intense filling of the left cardiac cavities and secondary opacification of the aorta from the left ventricle. Thus, the aorta is refilled

Operation does not come under consideration

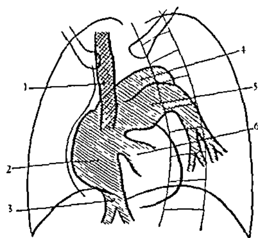


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aorta (arcus aortae sinister) is observed by analogy to the right sided aorta (arcus aortae dexter) with situs solitus (p 409). Diverse cardiac anomalies may occur with viscerum inversus totalis. Moreover, bronchiectasis is not rare (W. Neumann, Kartagener, Adams and Churchill, Zdansky) and indicates that some types of this lung lesion are congenital.

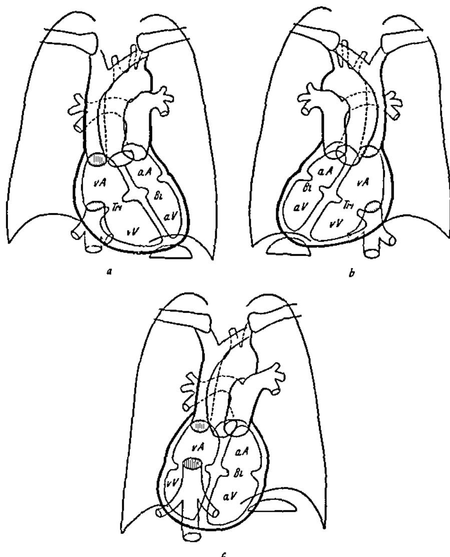


Fig. 223 —Schematic representation of various forms of dextrocardia (*a*) Normal situs (*b*) Situs viscerum inversus totalis. (*c*) Isolated dextrocardia without inversion of cardiac chambers ("dextroversion," Paltauf) (*d*) Isolated dextrocardia with inversion of the ventricles (*continued on page 311*)

Situs viscerum inversus partialis. Of much greater significance than total inversion is situs viscerum inversus partialis, irrespective of whether the thoracic or abdominal organs are involved. Often the relations are extremely complex and, for the most part, there are associated anomalies of the heart and vessels. Many of these anomalies are inevitable owing to the incongruous reciprocal relations between the

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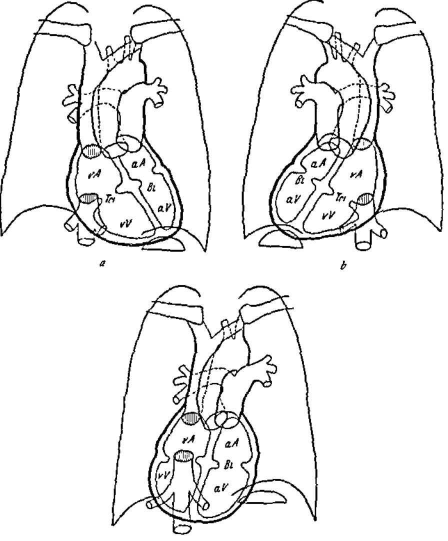


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(continued on page 311)

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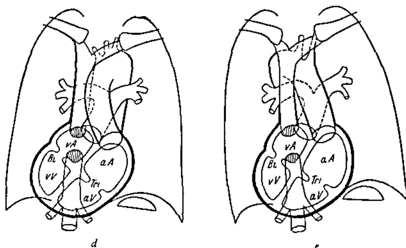
heart and the large abdominal vessels which must result in abnormal vascular connections or abnormal arrangements of the cardiac cavities to keep the circulation intact Some of these anomalies have vital significance and may ideally correct the abnormal anlage Other anomalies, on the contrary, are only single manifestations of a general developmental disturbance and may further complicate the situation by adding to the disturbance of oxygen supply to the organism

It seems didactically useful in describing the roentgen findings of the congenital positional anomalies of the heart to start with situs of the abdominal organs which can be detected by simple fluoroscopy. The position of the gastric bubble under the left and the liver shadow under the right diaphragm permits one to assume normal situs of the abdominal organs. The reversed position of the air bubble and the liver shadow justifies the assumption of situs inversus of the abdominal organs

If, with normal situs of the abdominal organs, the heart is located on the right, dextrocardia exists, if there is a situs inversus of the abdominal viscera but the heart lies on the left one speaks of levocardia

Dextrocardia may have various causes

1 There may be a congenital rotation of the normally developed heart around its long axis to the right. Paltauf calls this "dextroversion" (figs 223 and 224) The arrangement of the cardiac chambers to each other is normal although the long axis of the heart inclines from the left, posterior and above, to the right, anterior and below, by rotation, the left atrium forms much of the left border and the left ventricle contributes largely to the anterior cardiac wall, the right atrium is rotated



(*Fig 223 cont from p 310*)
and corrected transposition Aorta passes over the left bronchus (*e*) Like *d* but with right aorta Aa, arterial atrium, vA, venous atrium, aV, arterial ventricle, vV, venous ventricle, Bi, bicuspid ostium, Tri, tricuspid ostium

backward so that a large part of the right cardiac border is formed by the right ventricle The abnormal cardiac apex is directed to the right Slighter grades of dextroversion are called mesocardia (fig. 225). From the standpoint of circulatory physiology, dextroversion of the heart with normal situs of abdominal viscera lacks circulatory significance. Dextroversion is not an exact mirror image of the normal



FIG. 224 —Dextroversion of the heart with situs solitus of the heart and of the abdominal viscera (pseudositus viscerum partialis thoracalis). Male, 38 years old, without circulatory symptoms Both cardiac borders show a subdivision into two arcs (arrows) No pulmonary arc is present The aortic knob on left The left diaphragm is somewhat higher than the right and under it is the stomach On the originals, in the right lung field one could see the shadow of the horizontal interlobar fissure In other words, lobulation of the lungs was normal

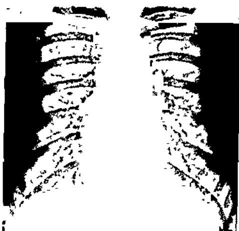


FIG. 225 —So-called "mesocardia" Female, 30 years old, without circulatory symptoms Electrocardiogram normal The heart is placed nearly median The right cardiac border is considerably elongated and a notch divides it into two arcs, the upper one displaying atrial pulsations, the lower, ventricular On the left cardiac border, only ventricular pulsations were noted A distinct pulmonary arc is present in the waist Left diaphragm is higher than the right

heart; no pulmonary arc is present within the excavation of the mid-right shadow corresponding to the cardiac waist, moreover, the right lower border is not strongly rounded like a normal arterial ventricle. Likewise, the location of the atrioventricular junctions, often merely a suggestion of notches on the two cardiac borders, are not mirror images (fig 224). Finally, the abnormal cardiac apex lacks the conic rounding of a true cardiac apex The right half of the diaphragm is lower than the

left; this indicates that the position of the heart and not the liver determines the physiologic differences in the heights of the two leaves of the diaphragm (Roesler).

2. A complete or partial inversion of the heart may cause dextrocardia. A complete inversion produces an exact mirror image of normal. In this way the heart loses its connections with the superior and inferior venae cavae and obtains blood from the extremities through veins which ordinarily serve other purposes or involute in the course of development (V. cardinalis sinistra, V. azygos, V. hemiazygos). Under these conditions complete cardiac inversion with normal situs of the abdominal organs loses significance. The roentgenogram is the exact mirror image of

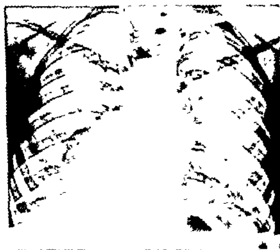


FIG. 226.—Dextrocardia with situs viscerum partialis thoracalis. Child, 4 years old, without subjective or objective circulatory disturbances. The cardiac shadow, of normal size, is placed toward the right. The atrioventricular junction on the right cardiac border is visible as a notch and lies fairly high above the diaphragm (at the level of the third anterior intercostal space). The root of the aorta lies at the left in front of the pulmonary artery. The vascular markings of the lungs are normal. The aortic arch passes over the left bronchus. An example of ideal correction of inversion of the ventricles by perfect transposition of the vessels.

normal. Very often, however, complete cardiac inversion with normal situs of the abdominal organs is associated with an aortic arch proceeding to the left or with grave anomalies.

In dextrocardia with partial inversion of the heart (figs. 223d and 226), the Vv. cavae open into an atrium lying on the right which communicates with its ventricle also lying on the right but through bicuspid valves, the arrangement of the papillary muscles and the conduction system characterize it as the real left ventricle. The pulmonary veins empty into an atrium lying on the left which communicates with a ventricle also lying on this side, this ventricle possesses, however, a tricuspid valve, a crista supraventricularis and an arrangement of papillary muscles and conduction system characterizing it as the real right ventricle. If the related aorta arose from the bicuspid chamber and the related pulmonary artery from the tricuspid, venous blood would immediately return to the systemic circulation through the

aorta and aerated blood would again return to the lungs; this would be incompatible with life. Life is possible only when the two vessels are displaced whereby the pulmonary artery arises from the functionally proper but anatomically improper venous bicuspid *ventricle lying on the right while the aorta departs from a functionally* proper but anatomically inappropriate arterial tricuspid ventricle lying on the left, Owing to this crossed transposition, the inversion of the ventricles is ideally corrected (v. Rokitansky) and the anomaly has no practical significance In the partial cardiac inversion with corrected transposition of the vessels, the aortic arch can proceed over the right or left bronchus (fig. 226)



FIG 227.—Dextroposition of heart resulting from a traumatic left-sided diaphragmatic hernia after a gunshot wound of the diaphragm Some fragments of the bullet (arrow) remain in left chest wall

Transposition corrected in this manner outwardly and roentgenologically resembles *dextroversion closely since the indentation of the left atrioventricular junction* is lower and that on the right higher and more distinct than in a mirror arrangement of the heart (fig. 223c and d). As in dextroversion, in dextrocardia from cardiac inversion, *the right diaphragm is lower than the left. The electrocardiogram* aids in the differentiation. In dextroversion there is no essential alteration while in dextrocardia from inversion lead I is the mirror image of the normal tracing, lead II resembles the ordinary *lead III*; and *lead III* resembles a *normal II* (*Holzmann, Taussig*).

Dextrocardia from rotation or inversion must be differentiated from acquired displacement of the normally arranged heart to the right, the latter is "dextroposi-

tion." It may be produced by congenital aplasia or hypoplasia of the right lung, *shrinking scars in the right lung or pleura, displacement of the heart by eventration* of the diaphragm, paralysis or upward displacement of the left diaphragm (fig. 227), left-sided space occupying processes (tumors, aneurysms, pleural effusions, pneumothorax), or some deformations of the bony thorax Pericarditis during fetal life might leave behind this type of displacement (Paltauf). Dextroposition causes a simple parallel shift of the cardiac axis or rotation of the heart around its sagittal

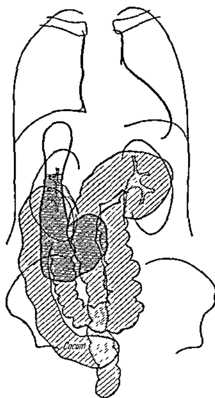


FIG 228 —Situs viscerum inversus partialis abdominalis So-called "levocardia" (Stomach on right, liver on left, left kidney higher than right, mesenterium commune) Female, 46 years old, with hemochromatosis Summary of films with barium-filled stomach and colon and an intravenous pyelogram

axis so that the long axis no longer proceeds from the right-above to the left-below but is almost vertical or runs from the left-above to the right-below The roentgen picture is never the exact mirror image of the normal heart It may closely resemble dextrocardia with a normal or inverse situs of the abdominal viscera Consequently, attention must be paid to the presence of differential diagnostic signs mentioned earlier A right pleural adhesion or shrinking lung scar, irregular notching of the right mediastinal shadow, adhesions in the right cardiodiaphragmatic angle, inspiratory distortion of *the mediastinum to the right, and inspiratory lifting of the right* cardiac border with the anterior chest wall, favor dextroposition rather than dextro-

cardia. Naturally a shrinking scar in the lung or pleura can be associated *with a true* dextrocardia.

Levocardia may also have different causes. First, it may be a heart laid down and developed in usual situs which may be considered completely inverse with respect to the normal situs of the abdominal viscera (fig. 228). Moreover, it is theoretically conceivable that levocardia could be combined with a partial inversion and corrected transposition of the great vessels. Finally, it is entirely possible that *levocardia could even result from left rotation of the heart in situs viscerum inversus* totalis, this rotation could be designated as levoversion by analogy with dextroversion Even without complicating cardiac malformations the differentiation of various forms of levocardia undoubtedly encounters difficulty. Levocardia with normal anlage of the heart yields completely normal cardiac findings (fig 228) since the abnormal venous connections are not usually detected in the roentgenogram and cardiac architecture is completely normal On the other hand, levocardia with partial inversion of the heart and corrected transposition of the great vessels cannot always be positively distinguished from the occasional occurrence of levocardia in a heart planned completely *inverse. Apparently no such cases have been described* in roentgenologic literature.

According to the congenital anlage of the heart, the following theoretic possibilities exist with normal and inverse situs of the abdominal viscera

	A Normal situs (situs solitus) of abdominal organs	
1 Heart to left	through situs solitus of the heart	Normal
2 Heart to left	through levoversion of the completely or partially inverse heart	Larval situs inversus partialis thoracalis
3 Heart to right	through complete inversion of heart	Situs inversus partialis thoracalis
4. Heart to right	with partial inversion of heart and corrected transposition of great vessels	Situs inversus partialis thoracalis
5 Heart to right	through dextroversion of heart planned in situs solitus	Pseudositus inversus partialis thoracalis
	B Situs inversus of abdominal viscera	
1 Heart to right	through complete inversion of the heart	Situs inversus totalis
2 *Heart to right*	*through dextroversion of heart* arranged in situs solitus	Pseudositus inversus totalis
3 Heart to left	through situs solitus of heart	Situs inversus partialis abdominalis
4 Heart to left	through levoversion of heart arranged inversely	Larval situs inversus totalis or pseudositus inversus partialis abdominalis

In all the possibilities listed, the aortic arch may proceed over the left or right bronchus. Basically, however, the malformations of the thoracic and abdominal organs leading to positional anomalies of the heart are prone to be associated with malformations or arrested developments of the heart and great vessels Consequently transposition of the great vessels is often associated with a septal defect, a Fallot or Eisenmenger complex, pulmonic or aortic stenosis, pseudotruncus communis, and so forth. Often the resultant roentgen pictures are extremely difficult to interpret Often angiocardiograms and cardiac catheterization are indispensible. The electrocardiogram plays an important role in distinguishing between inversion and rotation of the heart (see above).

In regard to the various possibilities listed above the following may be added.

With respect to the second type in the first division (A-2), levoversion of the inversely planned heart with normal situs of the abdominal viscera does not seem to have been observed roentgenologically. This larval form of partial thoracic situs inversus could be differentiated from normal situs of the chest organs only by careful analysis of the roentgenogram and by the electrocardiogram Favorable implantation of the great veins can create ideal circulatory conditions. As with every instance of partial inversion, naturally one must consider complicating cardiac malformations.

In regard to a situs inversus partialis thoracalis with complete inversion of the heart (A-3), the resultant roentgen picture may be the true mirror image of the normal heart in the absence of any other anomaly if the connections of the veins leaving the abdomen are established through other venous channels (p 313).

In partial inversion of the heart associated with corrected transposition of the great vessels (A-4) the roentgen picture of the heart (fig 226) is not a perfect mirror image (p 312). The aortic arch may proceed over the right or left main bronchus No circulatory signs exist because the anomaly is ideally corrected.

Dextroversion of the heart with normal situs of the chest and abdominal organs (A-5) can be called pseudositus inversus partialis thoracalis (figs. 223c and 224). When slight, it is known as mesocardia (fig 225). Since circulatory disturbances are absent, the congenital rotation of the heart to the right with normal situs of the abdominal viscera lacks any real significance.

Situs viscerum inversus totalis (B-1, fig. 223b) has no significance from the standpoint of circulation and presents the true mirror image of the normal heart The aortic arch can proceed over the right bronchus. Congenital anomalies of the heart which represent the mirror image of these anomalies with normal situs are fairly common but require no additional discussion at this time

Dextroversion of the heart otherwise normally developed with inversion of the abdominal organs (B-2) may simulate situs inversus totalis, one may speak of pseudositus inversus totalis No examples seem to have been reported As stated earlier, complete cardiac inversion can be distinguished roentgenologically and electrocardiographically from a rotated heart of normal development. Rotation of the heart never leads to a mirror image of the normal heart as happens with complete inversion Moreover, most cases of the latter kind would be complicated by some other anomaly

Situs inversus partialis abdominalis (B-3) with normal situs of the heart like situs inversus partialis thoracalis with cardiac inversion (A-3) can be corrected from the standpoint of circulation by abnormal or preformed veins Then, the roentgen image of the heart may be entirely normal The author observed a female, 46 years old (fig 228) with partial inversion of the abdominal organs and mesenterium commune but no circulatory disturbances The cardiac shadow was normal in position, size, and shape as well as in pulsations The pulmonary vascular markings were normal The aorta passed over the left bronchus Presumably the arrangement of the heart was essentially normal as with Geipel's patient who was examined only at necropsy, in the latter the hepatic veins emptied into a Vena hepatica communis while blood from the domain of the inferior vena cava reached the right atrium through the greatly dilated Vena azygos

As in situs inversus partialis thoracalis, the heart is often malformed so that severe circulatory disturbances develop Frequently interpretation is difficult. Precise analysis of the roentgenogram, electrocardiogram, angiocardiogram, and cardiac catheterization often is indispensible. Most patients with these grave anomalies die in early infancy although some cases, observed roentgenologically, have attained childhood or adult life (one case of Roesler at 6 years, two cases of Forgacs at 5 and 26 years, three cases of Zdansky at 3½, 8, and 19 years). Our first case, a male 19 years of age (fig 220a and b) had suffered from dyspnea and attacks of unconsciousness from early childhood. He had severe cyanosis and clubbed fingers The red blood cell count was 8.5 millions. Cardiac dullness was widened to the left The apical impulse was located in the sixth left intercostal space one finger beyond the midclavicular line. The first sound at the apex was loud and tambour-like. A rough systolic murmur was audible over Erb's point, occasionally a soft diastolic murmur was detected We noted the following (fig 220a), the cardiac shadow was considerably enlarged to left and right. The left cardiac border showed a rounded angle as is often seen in a Fallot anomaly. The gastric air bubble appeared under the right diaphragm, the liver shadow under the left The vascular band was wide. There was a pale, arcuate shadow, bending laterad in systole projecting below the aortic knob and in front of the initial part of the descending aorta The cardiac waist was normal as were the hilar shadows and lung markings Necropsy disclosed dextroposition of the aorta which rode a high ventricular septal defect and transposition of the hypoplastic pulmonary artery which departed from a small left ventricle The very small left atrium received a few pulmonary veins from a short common trunk This atrium led to a small left ventricle through a valve equipped with two leaflets. The right atrium and ventricle were large and formed almost the entire anterior surface of the heart, they communicated by a valve equipped with three leaflets The aortic arch passed over the left bronchus in a normal way. The hepatic veins opened directly at the diaphragmatic limit of the right atrium Substituting for the inferior vena cava, a persistent left cardinal vein ascended to the left of the descending aorta, crossed ventrad to the pulmonary artery and finally bent over the left bronchus to the right to empty *behind the ascending aorta into* the superior vena cava, the latter opened into the right atrium (fig 220b). This large vein received the left and right brachiocephalic veins as well as some trunks from the roots of the lungs The pale arcuate shadow visible below the aortic knob in the *roentgenogram corresponded to the left cardinal vein*

The second case, an 8 year old boy, was somewhat retarded in physical development, he had a marked bulge of the left anterior chest wall and a systolic murmur over the heart. On physical exertion, slight cyanosis appeared. The cardiac shadow had expanded considerably to left and right and its waist was filled by a conspicuous pulmonary arc. Fluoroscopy in the oblique positions showed that both halves of the heart, including the left atrium, were enlarged Systolic expansile pulsations were noted in the left hilar shadow. Angiocardiography revealed simultaneous filling of both enlarged atria and the large right ventricle. Then, the left ventricle opacified with simultaneous filling of both great vessels. The conus pulmonalis seemed narrow while the pulmonary artery was wide. The left heart and pulmonary artery retained

contrast material abnormally long A pulmonary stenosis and a persistent ductus arteriosus were assumed to be present.

The third patient, a girl 3½ years old (fig 229) had been cyanotic and dyspneic since birth, the fingers were clubbed and development was retarded A loud systolic murmur, transmitted to the lungs, had its maximum intensity over the pulmonary valve. The cardiac shadow was only slightly enlarged and showed a mitral configuration owing to prominence of the dilated pulmonary artery. The right ventricle was hypertrophied and moderately dilated. The accentuated hilar shadows and central vascular lung markings showed systolic expansile pulsations. The aorta was dextroposed The aortic arch passed over the left bronchus

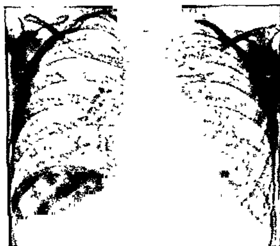


Fig. 229 —Situs inversus parietalis abdominalis Girl, 3½ years old, with acrocyanosis and clubbed fingers and toes Loud systolic murmur over the heart with maximum intensity over the pulmonic and transmission to the lungs The cardiac shadow is only slightly enlarged and has a mitral configuration owing to a flat bulge of the pulmonary arc The right ventricle is hypertrophied and moderately dilated The hilar shadows are enlarged and, like the increased vascular markings in the central lung fields, show systolic expansile pulsations The root of the aorta is displaced to the right The aortic arch proceeds normally over the left main bronchus

Forgacs described a child, 5 years old, who was also cyanotic and had clubbed fingers The heart was in normal position although widened to the right A loud, rough systolic murmur was audible with maximum intensity over the third left intercostal space The ascending aorta was displaced to the right and the arch passed over the right bronchus The author assumed a Fallot anomaly was present. An army officer, 25 years of age, had no circulatory symptoms and he was capable of exertion (Forgacs) A rough systolic murmur, audible over the second left intercostal space was conducted to the back The second pulmonic sound was accentuated The moderately enlarged cardiac shadow showed strong pulsations of a prominent conus pulmonalis and dilatation of the intrapulmonic vessels in which no pulsations were visible The ascending aorta seemed displaced to the right. In this

case as well as in the second one observed by Zdansky, an Eisenmenger complex could have been present.

Finally, *situs inversus totalis* (*B-4*) may awaken the *impression of situs inversus* partialis abdominalis owing to levoversion of the heart. Careful examination reveals, however, that the picture of an inverse heart, rotated to the left, never corresponds precisely to normal situs of the heart. Moreover the electrocardiogram permits a distinction Holzmann described such a case

—Chapter Five—

Diseases of the Pericardium

Except at a few places, the heart and roots of the great vessels are covered by pericardium This is so thin and clings to its contents so perfectly that the irregularities of the cardiac surface and vessel corona are outlined on its own surface *

By means of a re-enforcing fibroelastic layer, the tunica fibrosa, the parietal pericardium ensures fixation of the heart within the chest and offers some protection against overdistention The diaphragmatic part is joined firmly to the central tendon of the diaphragm, the mediastinal part is bound laterally and anteriorly to the loose connective tissue of the mediastinal pleura, this fixes the periardium to the anterior and posterior chest walls as well as to the roots of the lungs, finally the sternocostal part joins the anterior chest wall in the region of the "pleura-free triangle" by means of the Ligg sternopericardiaca and by loose connective tissue Behind, the outer leaf of pericardium borders on the connective tissue of the posterior mediastinum The esophagus lies in the median plane and is in apposition to the pericardium for several centimeters

The transition of the parietal into the visceral pericardium occurs anteriorly in a line which begins at the right edge of the superior vena cava above its orifice in the right atrium and continues to the left over the anterior edge of the ascending aorta (about 1 cm below the departure of the truncus brachiocephalicus) to the pulmonary artery, the latter is covered by the pericardium nearly down to the Ligamentum Botalli (fig 230a and b) In this way the roots of the aorta and pulmonary artery are mutually encircled by pericardium so that a passage develops, this is the transverse sinus of the pericardium, which separates these vessels from the superior vena cava

Posteriorly, the transition of the two layers of pericardium proceeds in two small sites of reflexion which run almost perpendicular to each other One proceeds craniocaudad and connects the posterior walls of the superior and inferior vena cava while the other runs horizontally over the posterior wall of the left atrium, skipping from one venous funnel to the other.

* The pericardial fat pad which frequently fills the left cardiodiaphragmatic angle (p 121) is formed by connective and fat tissue between the leaves of the pericardium and mediastinal pleura at their separation, it does not belong to the pericardium

Normally all these situations are invisible roentgenologically but awareness of them is important since they make many pathologic changes in the roentgen picture comprehensible.

The normal pericardium is not visible roentgenologically and dry pericarditis likewise cannot be detected directly. The lively pulsations of the cardiac shadow,

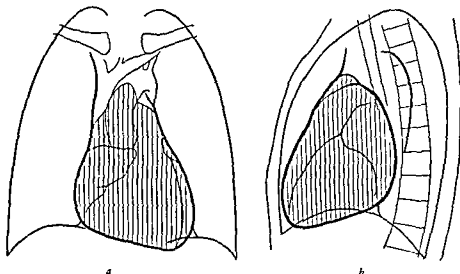


Fig 230 —Schematic drawing of the extent of the pericardium (a) Anterior view, (b) left lateral view (pericardium hatched)

occasionally observed (Brauer), *may be ascribed to the superficial shell of myocarditis* described by Virchow or to the diffuse myocarditis (p. 210) often present in pericarditis

1. *Pericardial Effusion*

A pericardial effusion which fills the entire pericardial cavity envelops the heart except at the dorsal sites of reflection and extends up to the origin of the great vessels

Attempts have been made to explain the distribution of small amounts of intra-*pericardial fluid by filling the pericardial cavity of cadavers* (v. Curschmann, Williamson, Alwens and Moog, Reinberg and Lindenbraten). Naturally the results of these observations should be transferred to the situation in vivo with caution The resistance which the working heart and pericardium opposes to the hydrostatic pressure of a pericardial effusion is entirely different from the resistance of dead organs and tissues toward artificially introduced fluid This is strongly emphasized by the fact that v. Curschmann was able to introduce 180 to 200 cc , Elias and Feller 400 cc. and at most 800 cc. into the pericardial cavity of cadavers while in life the cavity can hold 2000 cc. and more (v Curschmann), particularly when the fluid collects very gradually and inflammation loosens the tissues. Small amounts of fluid collect mainly in the diaphragmatic section, in the yielding left half of the pericardial cavity and in the furrows and angles at the base of the heart and between the

great vessels (v. Curschmann, Williamson). Predominant collection of fluid in the left half of the pericardial cavity may occasionally be clearly demonstrated by fluoroscopy after air is introduced into a cavity distended by fluid

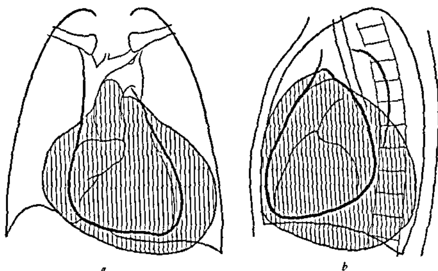


FIG. 231 —Schematic drawing of hydropericardium (a) Anterior view, (b) left lateral view (pericardium hatched)



FIG 232 —Large hydropericardium Large, nonpulsating cardiac shadow bulging to left and right with small vascular band Aorta of normal width Normal, light lung fields Pericardiocentesis yielded an exudate

Large effusions form a mantle of fluid over the heart. This mantle is not uniformly thick but collects largely to the left of the heart near the apex and diaphragm, less to the right and anterior, and least on the posterior cardiac wall (v. Curschmann) (fig. 231a and b).

Dietlen originally described the roentgen picture of a large pericardial effusion. The mediastinal shadow bulges particularly to the left, somewhat less to the right,

forward and backward; its contours below the vascular band often are nearly horizontal and reach the diaphragm in large, convexly curved arcs. (figs. 232 and 233a). Accordingly, the cardiac shadow on the left can extend to the lateral and posterior chest walls and may be broadly applied to them. Usually the cardiodiaphragmatic angle is acute in contrast to the obtuse Ebstein angle as determined by percussion. This contradiction is explained by the insertion of a small wedge of atelectatic lung between the liver and the expanded pericardium which escapes discovery by percussion. With very large pericardial effusions the angle may be obtuse.

Single arcs of the cardiac shadow vanish with large effusions when the heart, surrounded by fluid, is neither greatly enlarged nor pathologic in shape

The cardiovascular shadow is striking and has been compared to a gourd (Dietlen). Usually its transverse diameter exceeds the longitudinal With transverse projections this large mass is applied widely to the anterior chest wall and it also encroaches on the retrocardiac field Consequently the esophagus also sweeps backward in a wide arc and frequently to the right as well. Circumscribed displacement below the bifurcation like that seen in left atrial enlargement is also common.

The left diaphragm is displaced caudad by the weight of the fluid resting on it, often the gas bubble of the stomach is visibly indented from above Since the congested liver often displaces the right diaphragm cephalad, considerable differences may occur in the levels of the two domes.

The size of the cardiac shadow should not change during the Muller and Valsalva tests (Heckmann, Arendt)

Pulsations of the cardiac borders may be completely absent or almost imperceptible Pulsations may reappear on the left cardiac border when the upper body is inclined to the left (Heckmann)

In kymograms "vague, rounded contour movements" are described (Stumpf) although Heckmann observed an entirely straight line of the diastolic and systolic limbs of the curve The atrial movements vanish early, this has differential diagnostic importance from large nonfibrillating failing hearts in mitral disease in which atrial pulsations tend to be preserved along the right cardiac border.

In contrast to the small cardiac pulsations are the distinct aortic pulsations (Berner) However, they can be greatly reduced with large effusions which lead to a small stroke volume of the heart

With large pericardial effusions the lung fields differ in appearance Often with inflammatory effusions, they are perfectly clear and the hilar shadows are normal, that is, the lungs are free from congestion The effusion compresses the right atrium, the hepatic veins and the intrapericardial portion of the venae cavae (Elias and Feller) and consequently hampers the flow of blood into the heart, the pulmonary circulation obtains abnormally little blood and stasis develops in front of the right heart. The absence of pulmonary stasis stands in striking contrast to the size of the cardiac shadow; this has diagnostic significance It makes highly probable the differentiation between a decompensated aortic heart (hypertension, aortic valve lesion) and an inflammatory (rheumatic, bacterial, or uremic) collection of pericardial fluid. Naturally with uremic pericarditis the lung fields are often more or less hazy from renal pulmonary edema In contrast to exudates, transudates in the peri-

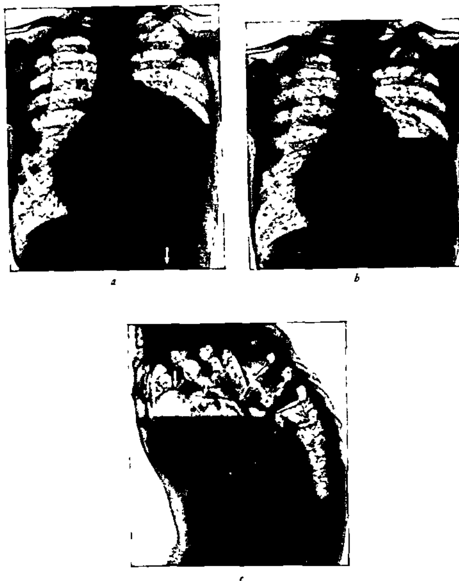


FIG 233 —Large pericardial effusion (a) Enormously enlarged, nonpulsating, undifferentiated cardiac shadow which bulges far to left and right below a short, narrow vascular band The gastric air bubble is indented from above (arrow) Normal vascular markings of lungs (b) Hydropneumopericardium after puncture in same case. One recognizes the unequal distribution of fluid in each half of the pericardial cavity and the attachment of the pericardium just below the aortic knob (c) Left lateral view of same case One sees the dome-like vault of the pericardium (arrows) which is superimposed only by the shadow of the aortic arch The barium-filled esophagus is projected into the left half of the pericardial cavity which bulges far posteriorly The esophagus indicates approximately the position of the posterior cardiac wall. In fluoroscopy the latter was perceptible within the shadow of the expanded left half of the pericardial cavity as a double contour.

cardial cavity resulting from stasis practically always are associated with pulmonary congestion. Although the roentgen findings, basically considered, do not permit definite statements about the nature of fluid in a cavity lined by serous membrane, nevertheless the roentgenologic features of the lung fields provide some information about the character of a pericardial effusion.

The heart cannot be outlined within a pericardial effusion. The intense central shadow sometimes interpreted as the heart proves to be a left atrium forming the border on the right (Dietlen), a *paravertebral abscess (Kloiber and Hochschild)*, a mediastinal tumor, mediastinal effusion, a pleural or pericardial adhesion (Amelung, W. Schmidt). Moreover, the lighter *marginal zone which the surrounding lung may* produce on the periphery of the cardiac shadow should not be confused with double contour formation (Assmann). Only small encapsulated collections of pericardial fluid located at one side of the heart may, owing to the thinner layer of fluid, be demarcated from the heart by virtue of the paler shadow In our experience, only one possibility of double contour has not been considered up to the present. With transverse projections, in the left half of the pericardial cavity, bulging far to the left and posteriorly, a darker shadow may occasionally be recognized which is produced by the right half of the pericardium containing less fluid than the left.

From this description one might think that the picture of a large pericardial effusion was characteristic and almost unequivocal. Nevertheless, this is not always true Assmann and Dietlen described mitral-tricuspid lesions in which the enormously enlarged cardiac shadow, bulging far to the left and right, was almost globular, nonpulsating, and closely resembled a large pericardial effusion Dietlen mentions a case in which only visibility of the enlarged left atrium within the right cardiac border and of the inferior vena cava within a low right cardiodiaphragmatic angle practically permitted the exclusion of an effusion On the other hand we mentioned earlier that in otherwise doubtful cases the absence of congestion of the lungs (clear lung fields, normal vascular markings) may be regarded a sign of probable hydropericardium.

If the recognition of a large effusion sometimes presents difficulties, the roentgen diagnosis of small and medium-sized collections fails fairly frequently. Opinions on the smallest amount of pericardial fluid demonstrable by x-ray vary widely. Traugott mentions a hemopericardium containing 700 cc. of blood which was not recognized as such clinically or roentgenologically. From a survey of all pericardial effusions confirmed by necropsy or paracentesis at the Massachusetts General Hospital from 1920 to 1930, White and Camp concluded that collections up to 500 cc. usually escape roentgenologic (and clinical) detection Even if such reports can be evaluated only with caution, since the effusion might have increased considerably between the examination and necropsy and since the serious condition of many patients or a coexistent pleural effusion permitted only a superficial study, still they indicate that the diagnosis or exclusion of such effusions belong among the most thankless tasks *of roentgenology.*

Although small and moderate effusions enlarge and alter the shape of the cardiac shadow, they may create unsurmountable difficulties in the differentiation from cardiac dilatation as may occur in a primary myocardial injury or a mitralized aortic

heart, with these dilatations the cardiac shadow often widens predominantly to the left, gradually to erase the different arcs of the heart and progressively to reduce all pulsations Moreover, splitting or double peaks of the marginal kymographic notches (Heckmann) occur not only with pericardial effusions but also in myocardial injuries In respect to differential diagnosis, the absence of pulmonary stasis and widening of the aorta is very significant in doubtful cases because they make a mitralized aortic heart highly improbable and favor a hydropericardium, naturally a myocardial lesion of inflammatory, anemic, or toxic nature cannot be excluded in this way In this connection we may recall the myxedema heart, concealed by a pericardial effusion which often surrounds a heart of normal size (fig 143a and b) Even lively pulsations do not preclude the presence of a hydropericardium

Certain alterations in the shape of the mediastinal shadow on change of body position have been regarded as signs of a pericardial effusion (Dietlen, Holmes, White, Kamenetsky and Rabinowitsch) They are supposed to result from gravitational displacement of the intrapericardial fluid Thus, the right cardiodiaphragmatic angle should become smaller in the recumbent position and larger on standing, moreover, the cephalic portion of the mediastinal shadow should become broader and the caudal portion smaller so that a more globular form is assumed on lying, particularly with elevation of the pelvis (fig. 234). Corresponding changes should also be seen in the left lateral position (v Curschmann). All these alterations of shape, produced by gravity, are inconclusive evidence of pericardial effusion because a relaxed heart may likewise change its shape with various postures (Heckmann) On the other hand, stability of cardiac shape despite change of posture does not preclude a pericardial effusion because a tense pericardium limits motion In medium-sized pericardial effusions inspiratory shift of the cardiac shadow to the right should be absent (Walldén) This sign is alleged to be trustworthy, however, only when diaphragmatic movements are adequate

Downward jutting of the diaphragmatic border of the heart (Brauer) and the absence of visible pulsations near the cardiac apex after air inflation of the stomach (Arcelin, Maragliano) also do not prove the presence of a hydropericardium

It is particularly difficult and usually impossible to decide how much an enlargement of the cardiac shadow in a decompensated valve or myocardial lesion depends upon myocardial dilatation or a coexisting hydropericardium This decision is rendered more difficult because the hydropericardium may resemble the shape of the heart it encloses and need not produce the classical gourd appearance particularly when the heart is enlarged and has a pathologic configuration (Zdansky) If a mitral lesion exists and the pulmonary arc protrudes, as a rule, the cardiac shape retains its mitral configuration despite an added pericardial effusion and usually the bulge of the pulmonary arc is preserved (fig 235) The pericardium has experienced corresponding tension and transformation owing to the enlarged, abnormal cardiac shape and the intrapericardial fluid follows the same distribution to recreate on a larger scale the form of the enclosed heart

These diagnostic difficulties and sources of deception have induced many to go so far as to ascribe any rapid, new change in the cardiac shadow to the appearance or absorption of a pericardial effusion, they would limit true changes in cardiac

volume at most to acute infectious or toxic myocardial injuries, severe anemia, or long lasting tachycardia (Dietlen) Undoubtedly *this generalization is too broad* because many volumetric alterations in the cardiac shadow are due expressly to *fluctuations of heart size*. For example, if a hypertensive heart or one with an aortic valve lesion widens predominantly to the left and signs of pulmonary congestion appear simultaneously, in short if signs of left heart failure develop, it is highly probable that the left heart has dilated even if this expansion of cardiac shadow later recedes. Likewise, predominant widening of the heart to the right and increased prominence of the conus and pulmonary artery, as frequently happens in mitral lesions and in cor pulmonale, favor an actual enlargement of the right heart. It is true, however, that often a pericardial effusion participates in transient enlargements of the cardiac shadow.

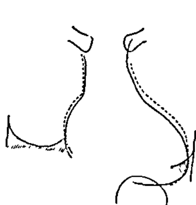


Fig 234

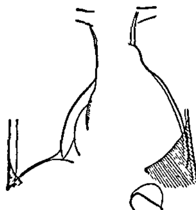


Fig 235

Fig. 234 — Transformation of cardiac shadow in the erect and recumbent position *in a patient* with pericardial effusion ——— Vertical orthodiagram, ---- horizontal orthodiagram Girl, 17 years old, with exudative pericarditis associated with polyserositis

Fig 235 —Pericardial effusion (established by paracentesis) in a case of mitral disease Female, 56 years old The shadow of the effusion suggests a mitral configuration (orthodiagram in heavy line) After absorption of the effusion, the large left atrium was visible on the right side of the heart (orthodiagram in thin line)

Angiocardiography permitted a real advance in the roentgen diagnosis of pericardial effusion (Williams and Steinberg, Levy and coworkers) Between the opacified cardiac cavities and the surrounding lungs there is an abnormally broad shadow corresponding to the fluid surrounding the heart The smallest volume of *fluid detectable in this way is unknown* Moreover, the value of angiocardiographic proof of a pericardial effusion is adversely affected by the fact that most patients coming under consideration are not suitable for this procedure

It is not uncommon, after partial absorption of an effusion, for residual exudate to remain encapsulated within pericardial adhesions These are more common on the right side than on the left They consist of a cavity surrounded by thick scars or a thin sac which *rests broadly on the heart and on the roots of the great vessels*. The enclosed fluid may be serofibrinous but more often it is fibrino-hemorrhagic and sometimes even purulent. Although often mentioned in the literature as "inflamma-

tory diverticuli of the pericardium" it seems advisable to limit the term "diverticulum" to a congenital or acquired herniation of the pericardial sac and to call the former encapsulated effusions (p. 331).

The roentgen picture of an encapsulated pericardial effusion varies greatly according to its position and size. The shadow has soft-tissue density; it is broadly applied to the heart and often to the roots of the vessels as well, it is ovoid or hemispherical with a border of simple or polygonal arcs and occasionally "overhangs" when the patient is erect. Usually it lacks pulsations unless they are transmitted ones Kienbock and Weiss once noted a calcareous deposit, the result of mural calcification, on the surface. Occasionally respiration and gravity change its shape. Thus, the shadow may flatten and lengthen when the diaphragm descends in inspiration or on transition from the recumbent to the erect posture it may become shorter and project farther into the lung fields during expiration and in the horizontal position. On standing, moreover, occasionally the "overhanging" appearance is created The lability of shape which Jaderholm and Jansson observed in so-called pericardial diverticulum depends upon the presence of a thin-walled sac filled with fluid.

Usually an encapsulated pericardial exudate does not change in size over the course of years, occasionally a gradual reduction or enlargement may be demonstrable

The roentgen diagnosis of an encapsulated effusion is never more than presumptive Encapsulated mediastinal effusions, diverse mediastinal tumors, primary and secondary tumors of the heart and pericardium, echinococcus cysts and even aneurysms of the ascending aorta (dell'Acqua and Freundlich) may present identical forms While lability of shape during respiration and from gravity tend to exclude the processes enumerated and favor an encapsulated effusion, doubt remains whether a pericardial effusion encapsulated by thin walls, a true pericardial diverticulum, or an encapsulated mediastinal collection is present.

2 *Pneumopericardium and Hydropneumopericardium*

Gas can collect in the pericardial cavity after the rupture of a tuberculous cavity, in connection with other inflammatory processes in the lungs accompanied by abscess or gangrene (Cowan, Harrington and Riddell) after rupture of an esophageal diverticulum, an esophageal, bronchial or gastric tumor, and even rupture of a gastric ulcer More commonly, intrapericardial collections of gas result from trauma. In these, the air may arise from the lungs, the esophagus, or directly from external wounds in the pericardium Observations of this type are made after bursting injuries of the lungs from chest contusion, after stab and gunshot wounds (Dietlen and Jenckel, Zdansky), after perforation of the esophagus by foreign bodies (Arens and Stewart), after accidental pericardial injury in thoracentesis or occasionally in the induction of pneumothorax (Saupe, Wegemer), and so forth At times, gas-forming micro-organisms within the pericardium seem responsible (Brauer, Brailsford).

True pneumopericardium is practically unknown, for usually the cavity contains a serofibrinous, purulent, hemorrhagic exudate, or blood in addition to gas (hydropneumopericardium). Hydropneumopericardium after paracentesis of an effusion with intentional (Wenckebach) or accidental introduction of air is most common. If the patient is examined in the erect position, the recognition of a hydropneumo-

pericardium is just as easy as the discovery of a pericardial effusion is difficult. It is characterized by a horizontal fluid level visible below the small collection of air within the arched distended domes of pericardium around the great vessels, in the anterior view there is often a large clear triangle *beside the pulmonary artery and a small one* beside the superior vena cava (fig 233b). With larger quantities of air the roots of the great vessels and the cardiac shadow are surrounded by a clear zone which is usually broader on the left than on the right; below, this stops at a horizontal fluid level and it is demarcated from the lungs by a thin, sharp line of pericardium lifted off the heart plus the adjacent pleura pericardiaca (fig. 143b). Usually the upper attachment of this line, 0.5 to 1.0 mm. in width, is a little thicker. Adhesions in the pericardium or mediastinal pleura, a coating of fibrin or neoplastic nodules on the inner surface of the pericardium, can make it much thicker (fig. 236). Then, its outlines may be vague and irregular.

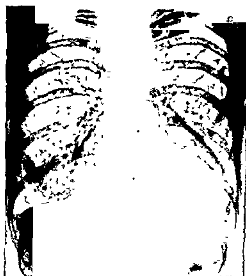


Fig 236.—Hydropneumopericardium after paracentesis of a hemorrhagic pericardial effusion Considerable thickening of the pericardium

The cardiac shadow displays very lively flapping pulsations resembling those seen *on the side of a pneumothorax.* They appear when direct application of encircling pericardium to the heart is eliminated and elastic support of the lungs is lost.

The fluid level universally surrounding the heart is thrown into constant waves by pulsations of the heart and by respiration. If only a little air and fluid are present the fluid level climbs to the left and in front of the heart with each diastolic enlargement of the ventricle, the smaller amount of fluid on the right executes much less movement. This suggests that essentially more unrest prevails in the left half of the *pericardial cavity than in the right,* a point of potential significance in the formation of pericardial scars and calcification The liveliness of the wave-like movements also depends upon physical factors; viscid pus moves more slowly than serous exudate. To ascertain whether the pericardial cavity is preserved elsewhere and that the fluid is freely moveable, the patient should be rotated through all planes of fluoroscopy and lateral positions employed as well.

3. *Pericardial Diverticulum*

Pericardial diverticula are circumscribed herniations of the pericardium of different sizes which are filled with fluid, the smallest range from the size of a pea or bean to larger ones, the size of a double fist. They favor the right half of the pericardial cavity and ordinarily are near the anterior wall of the thorax near a cardiodiaphragmatic angle. Often they communicate with the pericardial cavity only through a narrow stalk (Hart, v. Rokitansky, Bristowe and Schirmer). Usually the wall of the diverticulum is very thin and often it consists merely of a thin, relatively acellular layer of tissue which is covered only by an endothelial lining. The genesis of a pericardial diverticulum is not clear and probably not uniform. Some have thought a local zone, injured by inflammation, gradually protrudes owing to higher internal pressure. At present, it appears more probable that a congenital formation becomes manifest later in life (Mallory, Lambert). Possibly it concerns detached remnants of the ventral-parietal recesses of the pericardial coelum (His) as Lillie and coworkers suggest. Since the pericardial cavity develops from primitive lacunar clefts which gradually coalesce into a common cavity, pericardial diverticulum might also result from imperfect fusion of such spaces. In support of this conception is the fact that enclosed cystic formations without connection with the pericardial cavity exist in the same region and of the same structure, these cannot be regarded as outpouchings of the pericardial cavity (Mallory, Lambert, Bredford, Mahon and Grow). These pericardial coelomic cysts may be identical in nature with pericardial diverticulum. Radiologically they are indistinguishable from a pericardial diverticulum when they immediately adjoin the heart since it is impossible to determine whether or not the sac containing fluid communicates with the pericardial cavity.

Loculated effusions occur occasionally in adhesive pericarditis and must be distinguished from a pericardial diverticulum. They are encapsulated, often hemorrhagic, even purulent residual exudates. Although commonly called "inflammatory diverticulum" it is advisable to avoid this term (p. 329).

Only a few cases confirmed by necropsy have been published in roentgenologic literature.

The first case of this kind (Lenk) was interpreted as a pericardial tumor or a cardiac aneurysm. A 63 year old woman had luetic aortic regurgitation and chronic nephritis. Dullness to the right, anteriorly could not be separated from the heart. The roentgenogram revealed a round, soft-tissue shadow about the size of an apple on the right border of the enlarged heart, pulsating with the ventricle. At necropsy, the heart was large and a good-sized hydropericardium was present with a circumscribed outpouching of the pericardium to the right, about the size of a hen's egg. Siedler interpreted this formation as a circumscribed overdistension of a congenital weak point or one damaged by inflammation.

Also, in the second observation (Jaderholm), a pericardial diverticulum was not suspected. The patient, a woman 52 years old, had mitral disease. A round, sharply defined, nonpulsating, soft-tissue shadow about the size of an orange was located in the right cardiodiaphragmatic angle very close to the anterior chest wall. Posteriorly and above this shadow there was a line which gradually became thinner and followed the interlobar fissure, an encapsulated interlobar effusion was assumed. At necropsy, however, a thin-walled sac communicated with the pericardial cavity by means of an opening the size of a lead pencil, otherwise the pericardium was unaltered and contained only a small amount of fluid.

Most cases published as pericardial cysts are not actually examples of a diverticulum of the pericardium but closed coelomic cysts. Some have been established by operation (Lam, Mazer, Lambert, Cooper and coworkers, Lillie and coworkers,

Bishop and coworkers, Bates and Leaver, among others) The roentgen picture alone does not establish the diagnosis so that reports not confirmed by operation or necropsy must be considered questionable, this also holds for cases published by Kienbock and Weiss, Jansson, F. Ernst, Jaderholm, Arrilaga, Donovan and Taquini, Reitan, L. Haas, Donzelot, Bardin and Heim de Balsac, among others.

Pericardial diverticulum presents a homogenous soft-tissue shadow, sharply defined and simply articulated, resting broadly on the cardiac shadow. Corresponding *to its favorite site, this shadow is seen more often on the right than on the left, often* in a cardiophrenic angle and usually near the anterior chest wall. The shadow may display transmitted but not systolic expansile pulsations. Primary and secondary tumors of the mediastinum, pericardium, and heart, teratoma and dermoid cysts, as well as coelomic, bronchial, and echinococcus cysts, aneurysms of the aorta and of the heart, and pericardial or mediastinal encapsulated exudate, may produce very similar or identical pictures. If the shadow projects upward from the cephalic site of the pericardial reflection as in the cases of Jansson, Ernst, and Arrilaga, and distinctly in the case of Jaderholm, one may properly doubt the interpretation as a diverticulum. While the overhanging form as well as deformation of the shadow from gravity and respiration (Jansson) implies that the structure contains fluid, it does not *prove that a pericardial diverticulum is present. The same applies to calcification* of the wall. Consequently the diagnosis of a pericardial diverticulum is never established with certainty by x-ray.

4 Cicatricial Pericarditis and Mediastinopericarditis

If pericarditis heals by simply obliterating the pericardial cavity and without decided thickening of the membranes, cardiac work is practically unaffected and no circulatory disturbances or clinical signs indicate adherence of the pericardium. Examination of these patients yields nothing abnormal.

One speaks of pericardial adhesions only when scars thicken the pericardium. Volhard distinguished an internal and external variety. In the internal type, thick, *scarred, usually adherent pericardium encloses the heart In the external type* the adhesions extend to the mediastinal connective tissue (mediastinopericarditis), the endothoracic fascia, and the mediastinal pleura to cement the parietal pericardium to the anterior chest wall, the posterior and upper mediastinum, the spine, the diaphragm, or the lungs. *In the external type the pericardial layers usually adhere* to each other so that it is combined with the internal variety.

Neither kind of adhesive pericarditis necessarily produces circulatory disturbances Not rarely roentgen examination leads to their discovery and explains diverse, clinically obscure complaints such as pain in the chest, disturbances of rhythm and so forth.

Very often, however, severe circulatory disturbances occur and for awareness of them we are indebted primarily to Wenckebach, Ortner, Volhard, Schmieden, Sauerbruch, Brauer, Fischer, Rehn, Schur, and others.

According to Volhard, the internal variety primarily obstructs diastolic dilatation; the external hampers systolic contraction of the ventricles; both may finally lead to circulatory failure. Although no strict separation of the two forms is possible in respect to their mechanical and dynamic roles in circulatory failure

(Schur) this does not relieve us from the responsibility of attempting to locate the site of changes as far as possible in each case, the nature of anatomic alterations often determines the technical possibility and prospects of operative intervention which may save life External pericardial adhesions permit one to expect a successful result from precardiac thoracolysis (Brauer) while the demonstrable absence of extensive external adhesions and the presence of thick internal scars requires decortication of the heart which alone offers the possibility of surgical success. Therefore, roentgen examination is much more important than clinical signs like the "thoracic phenomena" (absent apical impulse, systolic retraction, diastolic sway of the chest, Broadbent's sign, Wenckebach's inspiratory retraction of the

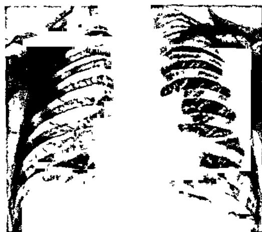


FIG 237 —Cicatricial mediastinopericarditis with diagnostic pneumoperitoneum Male, 18 years old, with ascites and widespread edema The cardiac shadow, of normal size, is deformed by cicatrices The pale shadow of the superior vena cava is widened considerably No mediastinal wandering of cardiac shadow in either lateral position No signs of pulmonary congestion In the base of the left lung a transverse atelectatic area An induced pneumoperitoneum demonstrated the absence of scars on the under surface of the diaphragm After resection of parts of some of the ribs on the right and the adjoining part of the sternum (Prof Denk) the ascites and edema permanently disappeared

lower sternum), pulsus paradoxus, inspiratory engorgement of the neck veins, hepatic stasis, ascites, edema and so forth These signs are equivocal and often only one or another is present Roentgen examination can provide valuable and often decisive diagnostic information which opens the prospect of release of the adhesions by a relatively minor operation at the correct place

Numerous signs of pericardial adhesions are assembled in roentgenologic literature. Although most of them are inconclusive, the coexistence of several and their relation to the clinical picture often facilitates the diagnosis, sometimes roentgen findings have overwhelming importance

The cardiac shadow may be normal in size (fig. 237) but often it is somewhat enlarged Extreme enlargement is uncommon but may be noted when a valvular lesion coexists or an encapsulated residual pericardial effusion persists

The shape of this shadow is subject to great differences Frequently this is

determined by the presence of a mitral or aortic lesion but very often the adhesive process changes it essentially. Pericardial scars, differing in thickness and location, can produce severe deformation of the cardiovascular complex by contraction and retraction so that frequently it becomes square or polygonal (figs. 237 and 238); often contraction stretches its borders (fig. 239). Commonly, notches or pointed extensions pull the mediastinal shadow into the lung fields or toward the anterior chest wall and often they become visible only with systolic contraction of the heart or deep inspiration. Occasionally the heart is drawn upward or to the side by these notches as each inspiration lifts the anterior chest wall.

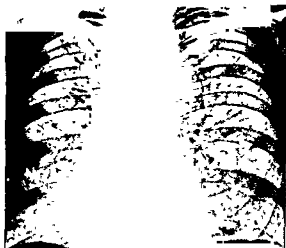


FIG 238 —Cicatricial pericarditis with extensive pericardial calcification and distortion of heart by right-sided pleuromediastinal adhesions Male, 65 years old, without circulatory disturbances and unaware of any previous illness

Naturally mediastinal, interlobar, and diaphragmatic pleural scars often participate in the deformation of the cardiovascular shadow; they pull the heart to one side and produce hazy, irregular demarcation of its shadow (figs 238 and 240). An unequivocal demarcation of the heart from these scars and from pleural effusions, on the whole, may be impossible.

Very commonly adhesions fill the cardiodiaphragmatic angle (Stuertz). Then, the median part of the diaphragm remains immobile or is elevated on deep inspiration while the lateral parts move distinctly downward (Achelis).

An upward systolic twitch of the left diaphragm (Schwarz, Dietlen) is not restricted to pericardial adhesions, not rarely, it is a normal phenomenon (Hitzenberger).

It should be reiterated that none of the signs just mentioned are conclusive evidence that pericardial adhesions exist, scars in the mediastinal pleura can produce most of them, scars may have no effect on the circulation or may lack any relation to any circulatory disturbances which happen to be present. In this connection one may recall that deforming pleuro-mediastinal scars and a contracting lung process



FIG 239 —Cicatricial pericarditis (accretio) Male, 64 years old, who suffered from increasing circulatory failure for two years The cardiac shadow is deformed by pleuromediastinal scars The cardiac waist is obliterated Pericardial calcification was detected over both halves of the heart Moderate pulmonary congestion The heart rose with the anterior chest wall in inspiration and showed no mediastinal wandering in the right and left lateral positions The shadow of the superior vena cava is widened decidedly



FIG 240 —Cicatricial pericarditis (accretio) Male, 30 years old, with ascites and congestion of liver and neck veins Distortion of right cardiac border by pleural scar which obscures the basal right lung field Calcification present on anterior wall of heart. Retrosternal field is narrow and obscured No pulsations along right cardiac border but large ones along the left Death after thoracolysis (necropsy)

(p 246) need not be accompanied by the least circulatory disturbance (Assmann, Dietlen) Often it is astonishing to see how even extreme pleural and pulmonary retraction fails to exert any real obstacle to the flow of blood into the heart although the venae cavae or branches of the pulmonary artery are greatly distorted and even kinked (figs. 163 and 164). Nevertheless, pleural scars can never be disregarded, in

the first place their extension to the pericardium and mediastinal tissues can throttle and distort the superior and inferior vena cava to produce inflow stasis (fig 241); in the second place they may indicate a coexisting pericardial scar; in the third place, *without involving the pericardium* and mediastinal connective tissue they may create severe circulatory disturbances such as cor pulmonale.

Soldering the heart on the anterior chest wall places the cardiac shadow in broad contact with the sternum and consequently narrows and obscures the retrosternal field, as is readily disclosed by lateral views. Occasionally the cardiac shadow *buckles on the posterior surface* of the sternum (Assmann) Then, deep inspiration neither widens nor lightens the retrosternal field since the adhesions lift the heart with the anterior chest wall.



FIG. 241.—Cicatricial pericarditis with right sided pleural scar Child, 15 years old, with ascites, congestion of liver, engorgement of neck veins and paradoxic pulse The roentgenogram merely shows a right sided pleural scar and considerable widening of the superior vena caval shadow (very faint on the reproduction) as an expression of inflow stasis Examination in right and left lateral positions showed no mediastinal wandering, indicating fixation of the heart in the chest

Extensions of the adhesions to the posterior mediastinum may also darken and narrow the retrocardiac field so that inspiratory widening and lightening is missed

These signs also should be appraised only with great care since the retrosternal as well as retrocardiac field normally varies greatly in depth; enlargement of the right ventricle or left atrium or pleural scars *can narrow and darken these spaces.* On the other hand, lateral views often reveal inspiratory fixation or retraction of the lower sternum as described by Gibson and Wenckebach (Dietlen) This last sign combined with darkening and narrowing to the posterior upper mediastinum and a vertical position of the heart is said to indicate the existence of posterior pericardial adhesions (Edens)

Occasionally in adhesive mediastinopericarditis we have observed single or [illegible] smaller and larger in the form of traction [illegible] *raction and kinking of the esophagus*, with [illegible] be ascribed to adhesions in the posterior

mediastinum (Sauerbruch); no cases showing this appear to have been reported in roentgenologic literature.

Pulsations of the cardiovascular shadow are often strikingly small or entirely absent when the heart is entombed in rigid scars. This observation is very compatible with the small stroke volume noted in many cases of pericardial adhesions. At times, however, pulsations of striking size are seen on the left cardiac border (Hosler, Cramer and Stehr). This seems prone to happen when scars fix the right heart while the left remains free (Fig 240). Then, the left ventricle is compelled to move toward the fixed right heart, also the slow flow of blood from the left atrium into the ventricle (Laurell) may play a role (p 103).

In roentgenkymograms the extremely small pulsations are characterized by lateral movements appearing at the end of diastole. This retardation of diastolic outward movement should reflect impaired diastolic dilatation by the ventricle enclosed in a cicatrix. Near pericardial calcifications (Cramer and Stehr) small, very brief systolic contractions of the ventricle are observed Likewise in concretio Berner found no pulsations or very limited ones in the zone of cicatrix as compared to surrounding areas. In inspiration the amplitude further diminished while double notches frequently appeared in expiration Berner believes this permits one to differentiate concretio from accretio by roentgenkymography since in the latter movements along the edge of the heart are unrestricted in every phase of respiration and in all positions of the body. We lack personal experience on the reliability of this sign

Recently McKusick demonstrated by electrokymography, alterations in the ventricular pulsations of patients with constrictive pericarditis which seem to be rather characteristic (fig 242). These changes are. 1) a V-shaped indentation contemporaneous with systole and joining the diastolic limb of the curve at an acute angle. Moreover, the latter is abnormally steep presumably because the ventricle is filled with abnormal rapidity by the atrium which stands under augmented pressure. 2) a diastolic plateau which begins prematurely, that is, before the end of diastole, and ends with the beginning of systolic ejection, passes into the descending systolic limb of the curve This plateau formation is an expression of the difficult filling of the ventricle which results in ventricular volume soon attaining its maximum and holding it until systolic emptying. 3) Absence of small strokes during the isometric phase of contraction and relaxation These signs are missed with tachycardia On the other hand a bradycardia may be accompanied by diastolic plateau formation and suggest a constrictive pericarditis. But in bradycardia the diastolic limb of the curve is not as steep as in constrictive pericarditis and the small strokes are preserved in the isometric period of contraction and relaxation

Special significance is attached to the observation of passive mobility of the heart on change of position and deep inspiration

It is certain that adhesions of the outer pericardium to surrounding structures can influence or completely abolish vertical shift of the heart However, if the heart is large, vertical shift is often lost even in the absence of any adhesions (Vaquez and Bordet).

Vaquez and Bordet believe that a study of the roentgenologic cardiac apex in the left lateral position and with deep inspiration helps in the diagnosis of pericardial

adhesions. Normally, in the left lateral position the apex moves to the left and in the erect position it moves caudad with the diaphragm in deep inspiration If there are firm adhesions between the apex and chest wall, *then the apex should fail to shift* to the left in the left lateral position even when the left ventricle arc becomes more rounded and projects, in the erect position and with deep inspiration it is lifted with the anterior chest wall If the cardiac apex is adherent to the chest wall as well as to the diaphragm, this would diminish or abolish respiratory shift of the apex as well as of the diaphragm. Finally, *if adherent to the diaphragm but not the chest wall, the*

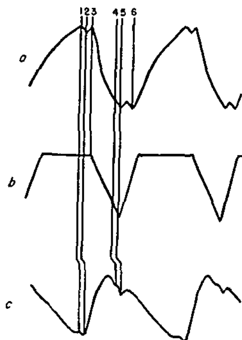


FIG. 242 —Ventricular electrokymogram of a constricting cicatricial pericarditis (After McKusick) The diastolic limb of the tracing ascends abnormally steep and ends prematurely The phase of ventricular filling forms a plateau The small excursions in the isometric phase of contraction,[1,2] and relaxation[5,6] are missing See text

(*a*) Normal ventricular electrokymogram (EKY), (*b*) ventricular electrokymogram in constrictive pericarditis, (*c*) carotid pulse

apex should participate in the respiratory movements of the diaphragm but not shift laterally in the left lateral position These rules of Vaquez and Bordet are supposed to be valid for the behavior of the apex except when a large aortic heart bulges to the left, then the apex tends to be *immobile even in the absence of adhesions* These rules do not mention the status of the cardiac base and the right heart border This somewhat schematic symptomatology has received criticism from Assmann, Achelis, Dietlen, and Zdansky. Certainly fixation of the cardiac apex to the anterior chest wall seems likely when the apex fails to shift in the left lateral position and it is lifted with the anterior chest wall by inspiration, the other signs, however, carry no great weight since pleural scars can also limit the apical mobility.

Of special importance in the diagnosis of pericardial adhesions is observation of

mobility and change of shape of the entire heart in the right and left lateral positions (p. 50). With external adhesions, both are limited or completely abolished if the heart is enclosed in scars and firmly anchored to surrounding structures On the other hand, in the pure internal variety of adhesions, only the shape of the heart is fixed since the heart enclosed by pericardial adhesions has lost its plasticity while its mobility within the chest is preserved.

The demonstration of the presence or absence of mobility and deformation of the heart is facilitated and improved by examination in the right and left lateral position with simultaneous deep breathing (Butler and Dana, Zdansky). Normally with deep inspiration in these positions the cardiac shadow moves toward the side turned upward (fig 31a and b) and changes its shape (p 50) The absence of this respiratory wandering in both lateral positions speaks strongly in favor of cardiac fixation by external pericardial adhesions Bilateral pleural scars, marked cardiac enlargement, a large hydropericardium, severe pulmonary congestion and pleural effusions, and the impossibility of breathing deeply also prevent mediastinal wandering in the two lateral positions (p 52). Unilateral pleural scars usually prevent wandering only in one lateral position while it is maintained in the other Owing to possible unilateral cardiac immobilization, examination in one lateral position is inadequate, the examination should always be performed in both positions. Only when wandering is absent or at least markedly reduced in both lateral positions should one assume, with the limitations cited above, that external pericardial adhesions exist.

Obviously this examination by no means covers all possibilities since circumscribed external pericardial scars may fail to immobilize the heart even when they cause severe circulatory disturbance by virtue of their special relation to the great vessels Consequently preservation of respiratory mediastinal wandering does not absolutely preclude the presence of pericardial adhesions

Moreover the shape of the heart need not be rigid in circumscribed pericardial adhesions On the other hand, the shape may be fixed under certain other conditions, thus the hypertrophic and enlarged heart offers considerable resistance to change of shape (p 51) Consequently maintenance of form is no more decisive for the diagnosis of pericardial adhesions than immobility of the cardiac shadow. In conjunction with the complete clinical picture, both signs have diagnostic value

Finally, calcium deposits in the pericardium should be mentioned as an important sign of pericardial adhesions (figs 238 and 243 to 246) Simmonds saw them first by x-ray in a cadaver heart and then Schwarz and Groedel observed them in living subjects Pericardial calcification is found in about one third of all patients with pericardial adhesions We have seen it many times

As a rule, pericardial calcification develops on the basis of a rheumatic, and more rarely of a tuberculous, pericarditis or hemopericardium One patient (Schwarz) received a blow in boxing twenty years before and one of our cases had a stab wound in the cardiac region four years earlier Calcification seems to require several years for development and gradually extends in the course of time (fig. 243a and b). In a patient who had an acute rheumatic fever nineteen years earlier Friedlander saw the calcification without a history of a preceding illness or injury, accordingly initiating pericardial lesion need not cause any symptoms to leave such residuals behind (fig 247)

Some cases have calcification so extensive that they embrace a large part of the heart and justify the name "armored heart" (Schwarz, Weil, Klason, Case, Scholz, v. Hecker, Friedlander, Assmann, Starck, Hessmann and Israelski, Cramer and Stehr, Freedman, and others). We observed three cases. The apex and the conus pulmonalis usually remain free of calcification (Muller) (figs 243 and 244).



a b

Fig 243.—Armored heart Male, 33 years old, with mitral lesion and resistant ascites The right cardiac border is markedly elongated and is divided into two arcs of which the upper belongs to the right atrium and the lower to the hypertrophied and dilated right ventricle No signs of pulmonary congestion Within the right cardiac border, the very large left atrium is seen as a darker nuclear shadow. In the left anterior oblique position (*a*) one sees, despite extensive calcification over both ventricles, the apex free of calcium At operation the two layers of pericardium were not adherent and the plate of calcium several millimeters in thickness, belonged exclusively to the outer layer of pericardium After partial removal of the calcium placques, the clinical findings remained unchanged During the next nine years, further extension of the calcification was noted

Usually the calcareous deposits are scattered over the cardiac surface as patches or map-like structures (fig. 245). They can easily escape observation when the rays *strike transversely or obliquely or when the shadows are projected into the heart* or spine They are, however, clearly demonstrable when the placque lies in the path of the ray, consequently the patient should always be rotated back and forth With *optimal passage of the ray the calcareous streaks vary in breadth* and are often nodular, they lie from one to several millimeters within the surface of the cardiac shadow and run parallel to it. Often they seem to project into the depth of the heart *but this is usually an illusion produced by part of the placque bending* toward the

intervention. A decision whether the calcification belongs to the visceral or parietal leaf of the pericardium is usually impossible. Once, v. Hecker was able to distin-





FIG 244 FIG 245

FIG 244 —Armored heart Male, 34 years old, with massive ascites and hepatic congestion Widespread pericardial calcification which left only the apex free (necropsy). (Left lateral view)

FIG 245 —Cicatricial pericarditis with map-like deposits of calcium in the pericardium Male, 53 years old, with massive ascites and hepatic congestion. No respiratory mediastinal wandering in right and left lateral positions (Left anterior oblique position)





a *b*

FIG 246 —Circular calcification of pericardium Acute polyarthritis eighteen years earlier No disturbances of circulation in this male, 54 years old (*a*) Left anterior oblique view, (*b*) left lateral view

guish a double calcium layer which he interpreted as separate calcification of the epi- and pericardium. In this connection it should be noted that calcification is not necessarily associated with adherence of the two layers of pericardium, a cleft can persist between them (fig. 243).

Calcifications are most common at the sites of pericardial reflection and where exudate remains longest owing to lesser cardiac movement or in consequence to furrows or unevenness of the cardiac surface (Müller). Rings of calcium may embrace the heart (fig. 246a and b). Once we saw a calcified placque in the sinus transversus pericardii, which extended to the right over the ascending aorta and to the



Fig 247 —Pericardial calcification at root of aorta and in sinus transversus pericardii Male, 38 years old, without circulatory disturbances or previous history of cardiac or pericardial disease

left as a bar corresponding to the sulcus coronarius ant. (fig. 247) Calcification is particularly common along the course of the sulcus coronarius (Grodel, Zehbe, Klason, Holzmann, Cramer and Stehr, Zdansky) the sternocostal and diaphragmatic surface of the heart (Klason, Horsch, Zdansky).

Calcifications in the left coronary sulcus are most easily recognized, in the anterior view, they usually begin along the left border a few millimeters within the arc of the left atrium and descend in a slight left-convex arc vertically in the cardiac shadow. Calcifications in the right coronary sulcus, on the contrary, are often invisible in the posteroanterior position and are found just to the right of the spine near the diaphragm The extent of calcification of the atrioventricular ring is seen best in the left anterior oblique position. It is precisely these calcifications in the coronary sulcus which often represent relatively insignificant incidental findings, they do not narrow the coronary arteries which pass through them as through tunnels (Hessmann and Israelski).

Even extensive calcification may cover large parts of the cardiac surface without causing symptoms or circulatory disturbances (Heimburger, Schmieden, v Fischer, Horsch, Hessmann and Israelski, Zdansky) (fig 238) Observations like these suggest that early calcification of a scar may oppose its shrinkage and therefore constriction of the heart.

Visible pulsations of the heart may be pronounced despite extensive pericardial calcification Occasionally the placques shift on each other during pulsation (Horsch)

Pericardial calcification should not be confused with calcified pleural scars in the anterior or posterior costomediastinal angle, with deposits of calcium in the lungs, with calcified cardiac thrombi, or calcification of the valves, of the annuli fibrosi, or of the heart wall The location of the calcareous deposit on the surface of the heart is diagnostically decisive, therefore careful fluoroscopy with rotation of the patient behind the screen is important.

The lung fields present variable pictures in patients with pericardial adhesions They can be perfectly clear and show normal vessel markings, particularly when pure inflow stasis results from an obstruction in front of the heart and when pleural scars are absent. Pleural effusions are frequently present in inflow stasis, then, the bases of one or both lung fields are obscured. Signs of pulmonary congestion are common when a mitral or aortic valve lesion coexists, when the heart is embedded in thick scars, or when the discharge of blood from the lungs is rendered difficult by a special location of the scar Pleural scars often obscure lung fields (fig. 237), particularly when they are edematous, a common event, or when they are actually very thick

5. *Tumors of the Pericardium*

Primary tumors of the pericardium (sarcoma, hemangioma, fibroma, lipoma) are rare Lenk described a case in which he interpreted a sharply defined, ovoid, rapidly growing mass on the right border of the heart as a malignant pericardial tumor A rhabdomyosarcoma of the pericardium (Bradley and Maxwell) caused massive enlargement of the entire cardiac shadow, and its right border had an irregular, nodular outline The lungs contained multiple round metastases At necropsy the tumor surrounded the entire heart and also penetrated the myocardium In a similar case (Steuer and Higley) the greatly enlarged cardiac shadow had a nodular border on all sides Jellen and Fisher saw a large solid teratoma in an infant, the enormous shadow could not be separated from the heart

Secondary tumors are much more common Naturally they do not differ essentially from primary ones They may be encountered as solitary or multiple protuberances on the cardiac surface Surface extension of the tumor can enlarge the cardiac shadow considerably and can reduce the pulsations or make them disappear

The effusion which often accompanies primary and secondary pericardial tumors may completely dominate the roentgen picture Behind every obscure pericardial effusion a tumor may be concealed

Pictures resembling tumors can result from pericardial or mediastinal encapsulated effusions, pericardial diverticulum, tumors of the heart or diaphragm, cardiac aneurysm, aneurysm or hematoma of the coronary arteries A diagnostic pneumothorax or pneumopericardium occasionally may assist in determining the relation-

ships of the shadow in question (Rosler), recourse to this diagnostic adjuvant is rather infrequent since the nature of the structure usually remains obscure even after its position is precisely determined.

A multilocular cyst filled with clear fluid and located over the apex in the parietal leaf of the pericardium was described by Yater. The convexly outlined, well defined projection over the apex could not be distinguished from a cardiac aneurysm Wright saw a cyst, the size of an apple, with a calcified wall in the parietal leaf of the pericardium in a male, 25 years of age, who had sustained a chest injury seven years earlier. The round shadow, outlined by calcium, rested on the cardiac waist and showed transmitted pulsations. It is not beyond the realm of possibility that this was a true pericardial diverticulum (p. 331).

—Chapter Six

The Lesser Circuit in Cardiovascular Diseases

The Lungs in Diseases of the Circulation

No roentgen examination of the heart is complete unless the lungs receive careful consideration Entirely apart from the demonstration of emphysema or extensive pulmonary or pleural cicatrices which have great importance in evaluating the heart, x-ray examination of the lungs offers a very instructive insight into the blood content of the pulmonary circulation, the amount of transudate in the lungs and the fluid content of the pleural cavities The examination affords an excellent possibility of detecting the presence of acute or chronic pulmonary congestion, insidiously developing pulmonary edema, a costal or interlobar effusion and sometimes the location of a pulmonary infarct

1 *Cardiac Pulmonary Stasis*

Arterial hypertension in the pulmonary circuit, the result of heightened resistance in the lungs from emphysema, kyphoscoliosis, pleural scars, or a primary pulmonary sclerosis, can produce demonstrable changes if the larger branches of the pulmonary artery dilate. In this way the hilar shadows enlarge and become thicker while the arteries radiating into the lung fields widen (Assmann) Often these vascular shadows show increased systolic-expansile (intrinsic) pulsations as evidence of greater tension in branches of the pulmonary artery caused by *abnormally high systolic pressure from contractions* of the hypertrophic right ventricle. Near the hilus, abnormal, large round shadows corresponding to the cross section of dilated vessels, often show distinct systolic-expansile pulsations Moreover the hilar shadows though enlarged are sharply defined and the lung markings are accentuated unless inflammatory processes complicate the picture The lung fields are clear or—as in emphysema—even abnormally translucent.

If pressure is increased in the pulmonary veins as well as in the pulmonary

arteries, in a mitral lesion for example, the vascular markings in the central portion of the lungs are even more pronounced (fig. 99) According to Sylla, engorgement of the pulmonary veins is expressed initially in the accentuation of the lung markings running from the left hilus to the apex of the left lung and later from the right hilus to the right pulmonary apex. The circumstance that the vascular markings are accentuated particularly in the central parts of the lungs occasioned Sylla to speak of a "central pulmonary congestion." In this stage of pulmonary engorgement, despite considerable enlargement of the hilar shadows, the lung markings are sharp and the lung fields are clear.

None of these alterations indicate cardiac decompensation, rather they express the heightened pressure in the arterial or venous limb of the pulmonary circulation. Thus, in emphysema or in a mitral lesion the increased vascular markings are the result of pulmonary arterial hypertension caused by the competent hypertrophic right ventricle, moreover, augmented venous markings in mitral lesions are considered, at least at first, merely the sign of increased pressure in the pulmonary veins, the consequence of diminished pressure gradient. Stasis in the capillary system and the transudation into the interstitial tissue and alveoli are still entirely absent. Strictly considered, no pulmonary congestion has occurred as yet.

With growing disproportion between the influx and discharge of blood, pulmonary stasis occurs. Then the picture changes by progressive dilatation of the large vessels, by capillary engorgement, and by transudation from the vessels into the parenchyma and interstitial tissue of the lung.

The hilar shadows undergo further enlargement, the lung markings become broader and more plentiful and a fine network, at times extending to the outermost periphery of the lung fields, appears between the thicker vascular shadows More important is the qualitative change in the lung structure More specifically, the vascular shadows lose their sharp contours; definition becomes vague and outlines blurred (fig 248). Often conglomerate shadows surround the massive hilar shadows which can be distinguished only with difficulty near the mediastinum Both lung fields become increasingly dark Cloud-shaped shadows appear in the bases of both lungs

The intensification and increase of lung structures as well as the diffuse darkening of the lung fields depends upon increasing engorgement of dilated pulmonary vessels including the capillaries and upon transudation into the pulmonary parenchyma. A very important contribution to the accentuation of lung markings and enlargement of the hilar shadows is made by the engorgement of the perivascular, peribronchial and pleural lymphatics, as well as the hilar and perihilar lymph nodes which are filled with transudate (Zdansky). Thus, with marked pulmonary congestion, the arteries, veins, and bronchi are often surrounded by greatly dilated lymph spaces filled with acellular fluid Moreover, the lymphatics of the interstitial tissues and pleura as well as the sinuses of the hilar and perihilar lymph nodes are crammed full of the same fluid Consequently, the bronchopulmonary nodes occasionally protrude as nodular shadows at the hili.

The vague demarcation and hazy appearance of the lung markings and of the hilar shadows deserves special explanation. Respiratory movements have importance for circulation of blood and lymph in the lungs. In pulmonary stasis the poorly

ventilated areas of the lung have increased discharge of transudate as a consequence of the slowing of the blood stream and diminished absorption of transudate as the result of slowing of the lymph stream. In this way transudate tends to collect in certain places just as has been known for a long time with hypostasis in the basal parts of the lungs. Wherever respiratory movements of the lung parenchyma are reduced owing to their connection with broader interstitium, the same event can happen (Zdansky). Around the hili and along the larger blood vessels and bronchi, particularly at their bifurcations, alveoli are filled with copious amounts of transudate and cells This tends to accentuate lung markings as well as contribute to their vague, faded appearance (Zdansky, Herzog)



FIG 248 —Pulmonary congestion in decompensated hypertensive patient The hilar shadows are enlarged considerably and the contours are faded The markings of the pulmonary vessels are increased but vague In general, the lung fields are cloudy

Apart from these streaks around the vessels and bronchi, the congested lung often contains partly indistinct and sharply outlined shadows of varying extent and number without recognizable connection to the physiologic interstices The smallest of these foci are pin-head in size, if they fill the lung very diffusely they produce the "spotted" appearance (fig 249) (Wierig, Fahr and Rosenhagen, Zdansky, Kadmy, and others). The appearance may simulate miliary tuberculosis, silicosis or a carcinosis.

All these focal condensations represent essentially circumscribed collections of transudate in the parenchyma with reduced respiratory movements The best known are the hypostases already mentioned, they are found as cloud-shaped shadows which may become confluent in the bases of the lungs Very often, however, indurations in the lungs cause circumscribed collections of transudate. On the basis of tuberculous or nonspecific indurations, large irregularly distributed foci of local transudation may suggest bronchopneumonic foci and infarcts, on the other hand

small, diffusely, scattered condensations of chronic brown induration of the lung may give rise to the previously mentioned stippled spots of transudation In the larger shadows atelectasis also plays a significant role (Zdansky) and obviously depends upon occlusion of small bronchi by swollen mucous membranes and their retained secretion (stasis catarrh).

According to the amount of transudation, the lungs show these types of condensation of varying size and more or less vague demarcation. Their size and structure may change as pulmonary stasis increases or recedes, with diminution of acute stasis and decreased transudation in the lungs these shadows may recede very

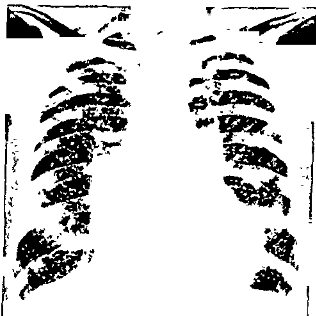


FIG 249 —Chronic pulmonary congestion of cardiac origin (mitral-aortic lesion) with fine stippling in the lung fields

rapidly in extent and number, the strands of lung markings become thinner, the hilar shadows become smaller and regain their sharp contours In place of the soft shadows, dense streaks and patches of condensation may now appear, representing collections of transudate and nests of heart failure cells. The dry lung of chronic congestion is clear, its markings are definitely increased and reticulated (Sylla), the vascular markings are thick but sharply outlined and the soft foci are absent.

Moreover, sections of the lung poorly ventilated owing to pleural scars and kyphoscoliosis often show pulmonary stasis. Copious accumulation of transudate in a section of lung enclosed by pleural scars may cause extensive, sometimes lobar, intense shadows suggestive of pneumonia but they vanish rapidly when the circulation improves (Zdansky). These observations are in keeping with the old clinical experience (Jürgensen) that pulmonary congestion appears earliest in the area of a pleural scar and stasis bronchitis is especially persistent in such areas

These unilateral collections of stasis fluid are due to the hampered removal of transudate as the result of diminished respiratory excursions and obliteration of the

lymphatics (Zdansky) Throttling of the pulmonary veins by mediastinal scars or kinking from pneumothorax, tumors and unilateral elevation of the diaphragm leading to unilateral pulmonary congestion (Teschendorf, Haubrich) is not definitely established and certainly is very rare if it exists at all

Unequal distribution of stasis transudate in the lungs could also result from regional diminished excretion of fluid Thus, in the vicinity of a circumscribed emphysema one may miss the signs of pulmonary congestion completely while the remaining parts of the lungs show abundant moisture. Obviously, emphysematous lungs with their poor vascularization discharge much less exudate than sections with normal circulation (Zdansky).

Lamellar atelectasis (Fleischner) in the form of small linear streaks, sharply outlined with optimal projection, are common in congested lungs (Pohl). They radiate like fingers from the hilus when a coexisting pleural effusion hampers ventilation in a lung already working under difficulty.

Pulmonary congestion which develops within a relatively short time, a common event in left heart decompensation from aortic valve lesions, hypertension, or after myocardial infarction, displays a picture somewhat different from that of chronic pulmonary congestion. In it the diffuse haziness or even focal shadows dominate over the accentuation of lung markings since there is more moisture of the lungs from edema fluid, less dilatation of the large vessels and less chronic induration

Pleural effusions develop in failure of the left heart and particularly of the right heart The picture cannot be described in this place. From a small effusion which just fills the costophrenic angle or a small mural collection, there is every transition to a huge effusion which obscures most of the lung The imbibition of pleural adhesions by edema must be distinguished from lamellar effusions, in the former, edema fluid is deposited as a jelly-like mass between the lung and chest wall and produces a small mural shadow. Lamellar effusions change in breadth during deep breathing while respiration does not affect the diameter of a mural adhesion filled with edema fluid (Zdansky).

It has been known for a long time that pleural effusions forming in the course of cardiac failure tend to appear in the right cavity (Bamberger, Neusser, Wintrich). Satke explains this phenomenon by the lower pressure in this pleural cavity, by compression of the Vena thoracica longitudinalis dextra by the dilated right atrium, pleural adhesions, periaortic scars (Maresch), or the traction of the sunken, large, heavy heart. According to Leendertz, transudate is excreted principally through the visceral pleura and absorbed mainly by the parietal pleura Since the surface of the trilobate right lung is larger than of the bilobate left lung, the preference for the right side in pleural effusions of cardiac origin would be explained At all events, right pleural effusion is so regular an event that in patients with an exclusive left hydrothorax, two possibilities must be entertained to explain the deviation from the rule. first, it is not a transudate but an exudate which has developed in connection with a left sided hypostatic pneumonia, pulmonary infarct, or post myocardial infarction pericarditis, second, adhesions obliterate the right pleural cavity and prevent the development of a hydrothorax on this side

Since chronic pulmonary congestion leads so often to pleural effusions and to adhesions, interlobar encapsulation of transudate is common In regard to the

resultant roentgenologic symptomatology, reference should be made to the thorough description of Fleischner. Very regularly in pulmonary congestion a streak in the middle of the right lung field indicates a thin layer of fluid in the interlobar fissure *or thickened interlobar pleura*.

Occasionally with chronic pulmonary congestion and particularly in mitral lesions, scattered, dense, pinhead to pea sized shadows, round, arborized, or rosette in shape are found, sometimes mainly in the bases. They represent spherical, nodular or arborized bony foci of lamellar structure in the alveoli and in the pulmonary interstices without regular connection to the vessels or bronchi (Gander). Cases of



FIG 250 —Nodular bone formation in chronic pulmonary congestion (necropsy). The jagged and branched calcium shadows are most clearly visible in the base of the left lung.

this kind were described by Salinger, Diehl and Kuhlmann, Pohl and Janker as well as by Gross We may add three cases which were diagnosed roentgenologically and confirmed by necropsy (fig 250). Their genesis is obscure An etiologic connection with cardiac stasis is at least very plausible (Salinger, Pohl, Gross). Two of our cases had a mitral lesion for many years and the third a congenital cardiac malformation, an abnormally small left ventricle had produced retrograde stasis in a greatly enlarged left atrium and in the lungs. These foci may gradually enlarge over the course of several years (Gross)

2. *Pulmonary Edema*

Acute pulmonary edema, with or without antecedent pulmonary congestion of cardiac origin, from profuse transudation of fluid into the lung parenchyma naturally is rarely investigated roentgenologically. If a case is occasionally encountered, the lung fields as a rule show marked, nonhomogenous cloud-like shadow masses.

More often subacute, gradually developing, pulmonary edema is observed in the course of nephritis, essential hypertension, aortic valve regurgitation, mitral stenosis, and the like. Its roentgenologic discovery is particularly important because clinically it often escapes detection for a long time owing to its central distribution and to its dominant or exclusive interstitial location which eludes auscultation or percussion. At first, near the hilus one sees strands and meshes of shadows gradually fade toward the periphery to lose their sharp contour (Coe and Otell, Zdansky, Bodart, Werkenthin, Jackson). Within this network which gradually extends, soft shadows appear and they may merge in larger shadows. In this way symmetric nonhomogenous linear streaks and foci sometimes extend like a butterfly from the hilus (fig 251)



Fig. 251



Fig 252

Fig 251 —Central pulmonary edema in uremia (necropsy) The middle sections of the lungs contain linear and large soft focal shadows.

Fig 252 —Diffuse pulmonary edema, predominantly parenchymatous, in uremia (necropsy) Faded, indistinct focal densities are conspicuous

and finally fill both lungs (fig 252) When receding, the foci vanish first while the linear shadows persist for a long time (fig. 253) before disappearing (Zdansky). The soft patches correspond to edema fluid in the parenchyma while the reticulated and linear densities depend upon edematous infiltration of the interstitial tissue. Naturally, parenchymatous and interstitial pulmonary edema should not be strictly separated, frequently they are combined and one form often passes into the other.

Pulmonary edema does not always involve both lungs equally. The reasons for the unequal distribution of this edema are uncertain When receding it is particularly persistent at the edges of the lobe When pleural scars hamper respiratory movements on one side, the edema often develops more fully on the opposite, better ventilated side (Zdansky) Hess observed the same thing in terminal pulmonary edema In pneumonia and lung tumors Hugenin saw the pulmonary edema appear precisely on the healthy side. In other words, pulmonary edema seems to behave opposite to pulmonary passive congestion, since the latter tends to involve poorly ventilated parts of the lungs more severely (p 348f)

At present we are beginning to understand these events which seem at first contradictory and, moreover, variable. Investigations by Liljestrand, v. Euler, Cournand, and others have shown the significance of oxygen tension in single sections of the lungs for their blood supply and the pressure relations in the pulmonary circulation. Consequently, one must assume that hypoxia which appears in the poorly ventilated sections of lungs leads to contraction of the vessels supplying these parts so that blood is diverted to well aerated divisions; this is a thoroughly appropriate reaction. The consequences of this fact have practical importance. If, for example, in acute glomerulonephritis one lung is poorly ventilated because it is encircled by pleural scars or indurations after a tuberculous or nonspecific process, even this lung may remain free from pulmonary edema while the other, normally ventilated lung may show profuse transudation. It is very probable that the reason for this phenomenon is reflex contraction of the pulmonary vessels on the altered side from diminished ventilation; most of the blood must flow to the well-ventilated lung and, owing to the existing tendency for transudation and acute rise of pressure in the venous limb of the pulmonary circuit, edema appears in this lung Not only unilateral pulmonary edema but likewise roentgenologically demonstrable focal and regional distribution of nephritic, central, and toxic infectious pulmonary edemas, under otherwise normal conditions may be ascribed to the unequal regional ventilation and circulation of the normal lung.

Differences in the ventilation and perfusion can promote, depress, or deflect to other areas the local excretion and absorption of transudate by reflex and mechanical means. These matters are extremely complicated and, in respect to details, diverse factors determine whether and at what places pulmonary transudate will collect. The pulmonary circulation is not a completely uniform, homogenous system. This situation prevails even under normal conditions because in the normal lung there are well ventilated and well perfused areas besides those with less ventilation and less perfusion. It is even more valid for lungs containing residues of former pulmonary and pleural disease, insignificant per se, like the scar of an old primary complex, an old hilar lesion, subsequent infiltrations, nonspecific indurations of old pleuritis. These conditions may create great differences in the behavior of pulmonary circulation and they may come to light roentgenologically in disturbances of lung circulation (Zdansky).

Often a bilateral pleural effusion is superimposed on pulmonary edema. It may be the only sign of impending edema in nephritis. Pleural adhesions naturally may prevent the effusion or may encapsulate it. Frequently the adhesions create shadows owing to imbibition with edema fluid (see above).

It may be difficult to distinguish central parenchymatous-interstitial pulmonary edema from pulmonary congestion of cardiac origin, indeed, it is impossible if the heart is pathologically enlarged or its shape makes a cardiac-pulmonary stasis likely. If, however, the cardiac shadow is not enlarged and the peripheral parts of the lung fields show normal vascular shadows, one can interpret, with a fair degree of certainty, densities in the central lung fields, usually symmetrical, as pulmonary edema.

The differentiation between central or peripheral, reflex, renal and allergic edema on the one side and inflammatory processes on the other, is not always possi-

ble. Particularly when influenza prevailed, the author repeatedly saw focal shadows whose interpretation offered great difficulties (fig. 254) in patients with valvular lesions (especially in mitral lesions) and with the appearance of fever and bloody sputum. They are distinguished from ordinary influenzal foci by their great number and widespread bilateral distribution. We might suggest that the coexistence of pulmonary stasis and an inflammatory process in the lung would accentuate the transudation into the lung tissue. It might involve a summation of inflammatory and cardiac edema.



FIG 253

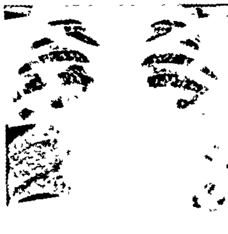


FIG 254

FIG 253 —Pulmonary edema, predominantly interstitial, in acute nephritis Opacities arranged in a linear manner are very characteristic of the interstitial form of pulmonary edema On the other hand, smaller patches of soft shadows are present and represent collections of transudate in the parenchyma In this case the pulmonary edema has developed more decidedly on the right than on the left side, perhaps owing to a left pleural cicatrix The edema vanished in three days

FIG 254 —Transient focal shadows in both lung fields during a febrile catarrhal process in a decompensated mitral lesion The shadows vanished within a few days

A picture very similar to central lung edema can occur in periarteritis nodosa Only a few instances of lung alterations have been demonstrated roentgenologically in this disease (Herrmann, Bodenstab, v. Conta, Becker, Postel and Laas, Svanberg) Most of them had lentil- to cherry-sized and even larger shadows, vaguely defined and occasionally confluent, in addition to accentuated linear shadows and reticulations Zdansky saw identical changes in a case of periarteritis nodosa, clinically definite but without necropsy. In one case (Postel and Laas) a parahilar shadow had fairly sharp outlines suggesting a tumor These focal and linear shadows may represent collateral inflammatory edema around periarteritic foci.

3. *Pulmonary Embolism*

Pulmonary embolism results from the entrance of a blood clot, tumor cells, fat, or air to a branch of the pulmonary artery. A vast majority of them occur in cardiac

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Often a bilateral pleural effusion is superimposed on pulmonary edema It may be the only sign of impending edema in nephritis. Pleural adhesions naturally may prevent the effusion or may encapsulate it. Frequently the adhesions create shadows owing to imbibition with edema fluid (see above)

It may be difficult to distinguish central parenchymatous-interstitial pulmonary edema from pulmonary congestion of cardiac origin, indeed, it is impossible if the heart is pathologically enlarged or its shape makes a cardiac-pulmonary stasis likely. If, however, the cardiac shadow is not enlarged and the peripheral parts of the lung fields show normal vascular shadows, one can interpret, with a fair degree of certainty, densities in the central lung fields, usually symmetrical, as pulmonary edema.

The differentiation between central or peripheral, reflex, renal and allergic edema on the one side and inflammatory processes on the other, is not always possi-

periphery, stands out by virtue of its abnormal clarity and sparse vascular markings. The normal vascular markings are interrupted directly at the border of this clear area (Westermark).

Corresponding to the heavy demand on the right heart, imposed by pulmonary embolism, frequently acute enlargement, often confined exclusively to the right heart, is noted Acute cor pulmonale may result (p 236).



Fig 255



Fig 256

Fig 255 —Pulmonary infarct Typical triangular shadow with the apex pointed toward the hilus and its base applied to the chest wall

Fig 256 —Marked widening of and increase of vascular markings in the lungs in polycythemia vera Male, 32 years old Erythrocytes 13 million, Sahli 166, leukocytes 4450, blood pressure, 180/80, splenomegaly.

4 *The Lungs in Polycythemia*

In polycythemia the lung markings often show striking, diffuse accentuation (Brednow). To some extent the picture recalls pulmonary congestion or a diffuse, fibroproductive hematogenous tuberculosis (fig. 256). The increase in markings is ascribed to the plethora, that is to the excessive filling of the vessels The increased number of erythrocytes lacks definite significance, for erythrocytosis without plethora is often unaccompanied by alterations of lung structure, moreover with reduction of the plethora, the accentuation of lung markings disappears

Brown and Giffin were unable to demonstrate any definite cardiac enlargement despite an increased volume of circulating blood Zdansky observed it only in patients with hypertension

patients with chronic passive congestion, and decompensation for these patients rather regularly develops a thrombosis of the deep veins of the pelvis and lower extremities.

Apart from large emboli which cause immediate death by occluding the mainstem of the pulmonary artery or a large branch, any embolus in the pulmonary artery, even if it is a small one, is a serious event. An embolus imposes a heavy burden on the right heart which frequently cannot be carried since often the myocardium is already damaged Just why embolic occlusion of some small branches can precipitate acute cardiac failure is not fully explained Reflex spasm of larger vascular fields in the lungs or heart is suspected (Scherf).

Embolic migration of a blood clot to the lungs leads to hemorrhagic infarction when pulmonary congestion coexists. The infarct is wedge shaped with its base turned toward the costal or interlobar pleura and its apex in the depth of the lobe With fresh lesions, the infarcted area is demarcated from normal air-containing lung or adjoining parenchyma unless this is filled with stasis transudate; soon, however, necrosis causes inflammatory exudation into the adjoining lung parenchyma or the pleura.

Corresponding to its characteristic shape, one would not expect the roentgenologic demonstration of the infarct to offer any particular difficulty This is, however, not true, for pulmonary infarcts are unequivocally recognized only in a minority of cases, this depends largely on coexisting pulmonary congestion which makes it difficult or impossible to outline the infarct shadow (Assmann, Rieder, Dietlen).

The shadow of a pulmonary infarct varies according to the plane of projection (Kohlmann). If the projection crosses the long dimension of the wedge almost perpendicularly, a triangular shadow is seen, if conditions are otherwise favorable: the base is directed toward the chest wall or an interlobar fissure and its apex is centrad (Fig. 255). If, on the contrary, the projection follows the long axis of the infarct, a rounded shadow, the projection of its base appears The resultant picture may simulate a metastatic tumor (Simonsen). Moreover the relatively characteristic picture is seen only with recent infarcts, later, vaguely outlined shadows cannot be definitely distinguished from hypostatic or pneumonic foci. This change in the infarct shadow depends upon local inflammatory processes. Furthermore, large lymph nodes are sometimes observed in the hilus and they may be connected with these inflammatory processes (Zdansky). Often the diaphragm moves less freely on the side of the infarction, perhaps owing to reflex immobilization from pleural irritation With upper lobe infarcts, elevation of the diaphragm on the affected side has been observed (Zweifel).

The infarct shadow may recede in the course of weeks and months and may leave behind an area of streaks or, sometimes, merely a linear shadow (Bohm and Kuhne, Zdansky, Hampton and Castleman).

If the infarct breaks down into an abscess, a central cavity soon appears, often containing a fluid level

If pulmonary congestion is absent, pulmonary embolism need not lead to a hemorrhagic infarct. The section of lung may become anemic since perfusion by a bronchial artery is insufficient. The involved section of lung, usually located at the

anterior view will be discussed later. At this juncture reference will be limited to changes of the vascular band produced by special spatial situations in the chest and particularly those created by abnormal levels of the diaphragm.

Elevation of the diaphragm (fig. 257) lifts the pillars of the aorta, that is, the beginning of the aorta and the hiatus aorticus. The resultant upward shift of the entire aortic loop is expressed roentgenologically by elevation of the aortic knob which may reach the level of the clavicle and even the jugulum. For anatomic reasons the cephalic shift of the aortic crest naturally lags behind the shift of the aortic pillars so that the limbs of the aorta bend sidewise. Now the ascending aorta extends farther to the right and may appear on the right border of the vascular band, replacing the superior vena cava. The aorta bulges into the lung field in a strong convex arc and is elongated at the cost of the right border of the cardiac shadow, finally it may project farther to the right than this border The descending aorta shows practically

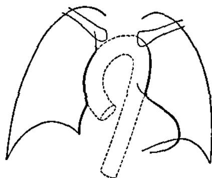


FIG 257.—Course of thoracic aorta with elevation of diaphragm By elevation of aortic ostium and of hiatus aorticus, both ascending and descending limbs of aortic loop move away from each other to widen and deform the vascular band

the same alterations As a strong convex arc it bulges into the left lung field, it may be widely exposed in the cardiac waist and can often be followed within the cardiac shadow to the diaphragm

The aortic knob can appear only when the distal division of the arch has an approximately sagittal course, since the arch proceeds obliquely through the thorax, the knob vanishes and in its place the arcuate transition into the descending aorta becomes visible

The opposite alterations in the course of the aorta are encountered from descent of the diaphragm If the aortic pillars move downward, both limbs of the aortic loop experience traction since the arch can follow the descent of the bases only to a limited extent. Although the aortic arch moves a little from the upper thoracic aperture, narrowing of the vascular band is much more striking Since the ascending and descending aorta bend inward the vascular band becomes narrower The right edge of the vascular band, formed by the superior vena cava, rises almost perpendicular, on the left side, at most, a very short, straight section of descending aorta is seen in the angle between the aortic knob and cardiac waist

The alterations in the course of the aorta which result from deformities of the thorax were discussed *on page 249*

Chapter Seven

The Aorta and Its Pathologic Alterations

I. The Normal Thoracic Aorta

Examination of the aorta is among the most rewarding tasks of roentgen diagnosis since clinical detection of diffuse and circumscribed aortic alterations is often difficult or impossible while roentgen examination often yields excellent information about the width, course, and gross anatomic status of the thoracic aorta

In discussing the normal cardiovascular shadow, reference was made to the extent that single sections of the aorta are visible in various planes of study. Only the most essential points will be repeated, the rest will be found on pages 21ff and pages 43ff.

The anterior view (fig. 12a and b) ordinarily reveals only the left edge of the distal section of the arch and the initial portion of the descending aorta as the aortic knob, the visible part of the descending aorta *descends almost vertically* from the knob toward the pulmonary arc and converges *slightly toward the midline*

Usually in anterior views the ascending aorta does not form the border since, on the right, the superior vena cava ordinarily reaches farther to the right. Only on occasion is the ascending aorta recognized as a darker shadow curved convexly to the right within the right edge of the vascular band.

With increasing age, the instances in which the ascending aorta projects to the right beyond the superior vena cava to form the border steadily increase. Then the *superior vena cava and the right innominate vein, as paler shadows, curve concavely to the right below the clavicle from the denser aortic shadow.* The extension of the ascending aorta beyond the superior vena cava is produced by aortic elongation and widening and by elevation of the diaphragm; these happen more frequently with advancing age.

In young children a projecting aortic knob (fig. 36) is often missed (Kreuzfuchs) the aortic arch, owing to the relatively great depth of the child thorax and the accentuated right position of the trachea proceeds more obliquely than in the adult. If these infantile relations persist, the aortic knob also fails to develop in the adult

The effects of pathologic elongation and dilatation of the thoracic aorta on the

anterior view will be discussed later. At this juncture reference will be limited to changes of the vascular band produced by special spatial situations in the chest and particularly those created by abnormal levels of the diaphragm.

Elevation of the diaphragm (fig. 257) lifts the pillars of the aorta, that is, the beginning of the aorta and the hiatus aorticus. The resultant upward shift of the entire aortic loop is expressed roentgenologically by elevation of the aortic knob which may reach the level of the clavicle and even the jugulum. For anatomic reasons the cephalic shift of the aortic crest naturally lags behind the shift of the aortic pillars so that the limbs of the aorta bend sidewise. Now the ascending aorta extends farther to the right and may appear on the right border of the vascular band, replacing the superior vena cava The aorta bulges into the lung field in a strong convex arc and is elongated at the cost of the right border of the cardiac shadow, finally it may project farther to the right than this border. The descending aorta shows practically



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The alterations in the course of the aorta which result from deformities of the thorax were discussed on page 249

Shrinking processes in the right lung and right-sided mediastinal pleural scars often pull the ascending aorta to the right so that it projects more markedly and may bend into a sharp arc. These pleural and pulmonary alterations merit special attention to avoid the erroneous assumption of dilatation of the ascending aorta (fig. 165).

The view of the aorta in the right anterior oblique position changes with the angle of rotation The ascending and descending aorta most nearly overlap with a rotation of about 30 degrees (Frik), thereby the right border of the vascular band tends to be projected into the dark shadow of the spine, making demarcation difficult. With greater rotation the descending aorta is projected more and more into the retrocardiac space

With a rotation of about 45 to 60 degrees (fig 17a and b) the anterior border of the ascending aorta comes into view as a short section above the flat pulmonary arc, but craniad it soon vanishes again within the pale shadow of the left brachiocephalic vessels which bend to the left above. Not rarely, however, one may follow the proximal portion of the arch to the clear tracheal band.

The posterior border of a normal or moderately dilated ascending aorta cannot be outlined with certainty since it is superimposed by the superior vena cava The latter forms the real right edge of the vascular band (p. 32) The posterior wall of the ascending aorta is not represented by a line dividing the left darker and the right lighter part of the vascular band as Vaquez and Bordet as well as Lippmann and Quiring believed This bipartite division of the vascular band is produced by the left main bronchus (fig. 19a and b) The posterior wall of the aorta can be outlined only when the vessel is dilated or its wall is visible owing to calcium deposits. Contrary to the opinion of Vaquez and Bordet and of Lippmann and Quiring a normal aorta is not accessible to exact anatomic measurement in this position. This would be possible only if the shadow of the superior vena cava surpasses the aorta exactly as far as the left main bronchus is projected into this vessel shadow (Assmann) To be sure, in many cases this actually happens (fig 261).

Normally the arch of the aorta cannot be followed since it is effaced by the clear tracheal band and furthermore cannot be outlined against the shadows of the brachiocephalic vessels and other soft tissues of the superior mediastinum.

Of the descending aorta only the ventral part is ordinarily recognized as a pale shadow descending to the left of the spine in the retrocardiac space, gradually to widen caudad

The thoracic aorta can be surveyed much better in the left than in the right anterior oblique position (fig 25a and b) since with this projection, all its parts are about equidistant from the screen.

Since the initial portion of the aorta is covered by the pulmonary artery, it is not demonstrable in this position The anterior border of the upper ascending division, however, is exposed for a fairly long stretch at the right border of the vascular band since the superior vena cava has already disappeared behind the ascending aorta when the patient is slightly rotated to the right (p. 41). Here again, demarcation of the posterior wall of the ascending aorta meets with difficulties owing to superimposition by the shadow of the right pulmonary artery

The proximal section of the aortic arch can be distinguished neither against the

brachiocephalic vessels and the soft tissues of the mediastinum nor within the clear band of the trachea Regularly, however, beyond the trachea the upper limit of the distal section of the arch and the descending aorta is visible within the vertebral shadow Moreover the anterior border of the descending aorta tends to be visible in the retrocardiac space as a pale shadow below the left bronchus although this varies individually and to different degrees depending upon the angle of rotation All these relationships were described in detail on page 41ff

Despite very fragmentary definition of the aortic loop, under normal conditions, examination in the left anterior oblique position offers valuable insight into the gross anatomic features of the thoracic aorta The anterior border of the ascending aorta, the upper edge of the distal segment of the arch and the posterior edge of the descending aorta can be followed with great regularity, consequently an excellent idea of the course of the loop is secured In this projection one can see how the ascending aorta courses straight or in a flat right convex arc obliquely to the left, passing into the strongly right-convex section of the arch and extending beyond the trachea as the descending limb, this continues as a flat curve, convex to the left with its dorsal section in the spinal shadow Diffuse elongation as well as diffuse or circumscribed dilatation of the thoracic aorta are revealed by deviations from this normal course This has great diagnostic importance and will receive further notice below.

It may be noted in passing that elevation of the diaphragm deforms the aortic loop and causes marked bulging of the ascending and descending aorta, so that the right edge of the vascular band pursues a convex course to the right, and the descending portion, visible within the spine, bulges more roundly to the left just as one is accustomed to see with diffuse aortic dilatation (p. 375)

In aged individuals the entire aortic loop can often be surveyed so that its diameter may be measured at any site selected In itself this would not be pathologic

II. Measurement of the Aorta

Numerous endeavours have been made to measure the aorta as exactly as possible.

Groedel introduced the conception of a transverse diameter of the aortic shadow (AT) By analogy to the transverse cardiac diameter, he tried to express the transverse diameter of the aorta as the sum of the right and left median distances (AMr + AMl) of the vascular band (fig 258) In normal adults, AT varied between 4 3 and 6 7 cm $\frac{AT}{2}$ was supposed to correspond approximately to the diameter of the ascending aorta Groedel also reported an "aortic length" (AL) In constructing it, plumb lines were dropped from the vertex of the aortic knob and the right cardiovascular angle to the midline and the distances between the points of intersection of these lines were measured on the midline (fig 258) In normal adults, AL was approximately 7 3 cm Zehbe and v. Teubern determined as "aortic breadth," the distance in teleroentgenograms or vertical orthodiagrams between the angle where the aortic knob meets the pulmonary arc and the point opposite on the right edge of the vascular band (fig 259). According to Zehbe the average values calculated for various ages varied between 4 6 and 5 9 cm, v Teubern considers them between 3 5 and 6 0 cm He also considers the distance of the right cardiovascular angle from the most remote point on the aortic knob, aortic length, for the adult male this measures approximately 8 7 cm Like Groedel, Vaquez and Bordet measured the transverse diameter of the vascular band, in horizontal ortho-

diagrams they found it between 4 0 and 8.5 cm, but they stress that the breadth of the vascular band as a measure of aortic diameter is influenced by the fact that the superior vena cava frequently forms the right border of the band

Actually these measurements have very slight value in deciding the length and width of the aorta This does not depend solely on the fact that the right border of the band is usually formed by the superior vena cava instead of the aorta. It is primarily because aortic dimensions are variably affected by spatial conditions existing in the chest (Assmann, Holzmann). Elevation of the diaphragm may widen the vascular band to simulate aortic dilatation, descent of the diaphragm reduces the width of the band making a dilated aorta look normal if the measurements just mentioned are performed alone. Moreover, asymmetry of the thorax (scoliosis), retrosternal

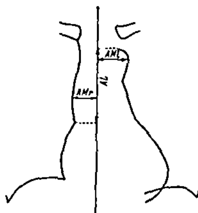


Fig 258

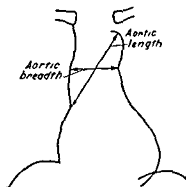


Fig 259

Fig 258 —Measurement of aorta by Groedel's method

Fig 259 —Measurement of aorta by the method of Zehbe and v Teubern

goiter and pulmonary and pleural retraction, especially on the right side may be sources of error

Despite the fact that the width of the vascular band depends upon factors unrelated to the status of the aorta, exact notice of its form is important Nevertheless alteration of intrathoracic spatial conditions should receive careful consideration, for they may produce decided changes in the course of the vessel.

Holzknecht was the first to attempt to measure the diameter of the ascending aorta He rotated the patient to the left until the ascending aorta overlapped the descending However, this measurement meets with difficulty because with this rotation, which amounts to 20 to 30 degrees, the right boundary of the aortic shadow is projected into the dark spinal shadow and cannot be unequivocally outlined

Vaquez and Bordet and Lippmann and Quiring attempted to measure the diameter of the ascending aorta by further rotation to the left (fig. 260). Their measurement is based upon the assumption that the left darker part of the vascular band corresponds to the ascending aorta while the right lighter section is formed by the superior vena cava to the extent of its projection over the aorta on the right. This assumption is unreliable (p. 34). Actually the clearer part is produced by projection of the left main bronchus into the vascular band (fig 19a and b) (Frik). Conse-

quently the anatomic basis for this measurement is untenable. Correct values are provided only when the left bronchus is projected into the vascular band up to the place where the superior vena cava extends over the ascending aorta to the right (fig. 261), for this, no absolute assurance exists. Since, however, this situation prevails in a large number of cases (Reich), under otherwise favorable conditions, the resultant values agree very well with predicted ones. As normal values for middle aged adults of average size and normal development, Zdansky found 2.5 to 3.0 cm These figures can be evaluated only when they concur with the figures obtained by the Kreuzfuchs method (p 363) and with the impression one receives about the aorta with sagittal projection and in the left anterior oblique position

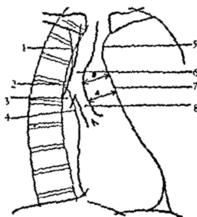


Fig 260 —Measurement of aortic diameter by the method of Vaquez and Bordet as well as by Lippmann and Quiring (A) and according to Assmann and de Abreu (B) in the right anterior oblique position

1 V brachiocephalica (anonyma) dext
2 V azygos
3 Right bronchus
4 V cava sup
5 Left brachiocephalic vessels
6. Trachea
7. Anterior boundary of ascending aorta
8 Left bronchus

Conditions are more favorable for measuring the proximal section of the arch (fig. 260) Usually the right posterior boundary of the arch is contrasted well against the left tracheobronchial angle. At this place the aorta lies directly on the angle which is usually readily visualized so that it is possible to secure an essentially exact orthodiagraphic measurement (Assmann, de Abreu) For technical reasons this measurement is not always possible. If the place on the left border of the vascular band opposite the left tracheobronchial angle is no longer aorta but, as often, the brachiocephalic vessels bending to the left and upward, measurement is feasible only when the aorta, owing to its greater density, can be demarcated within these vessels and soft tissue shadows Assmann pointed to an error which can become greater when the point on the left border of the aortic shadow at the level of the tracheobronchial angle is not the point where the aorta crosses the angle, this occurs when the elongated vessel is somewhat tortuous. Ordinarily these errors can be eliminated by making the measurement at the degree of rotation at which the

distance from the left border of the aorta to the tracheobronchial angle is smallest Such measurements are fairly exact for the diameter of the proximal section of the arch In adults of normal build and healthy from the standpoint of circulation, they

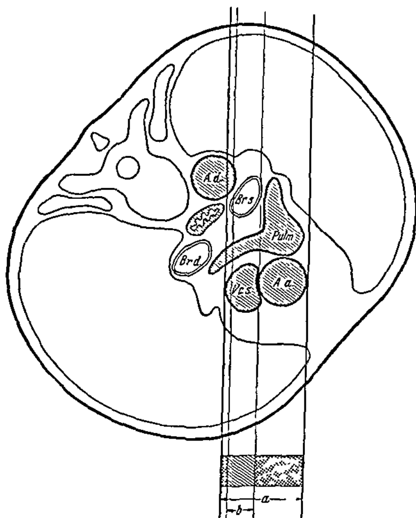


Fig. 261 —Measurement of the diameter of ascending aorta in the right anterior oblique position The width of the vascular band (*a*) at the level of the bifurcation of the pulmonary artery is formed by the shadow of the ascending aorta and the superior vena cava By virtue of the left bronchus, the vascular band experiences partial illumination (*b*) the left boundary of which, in an ideal case, coincides with the right boundary of the aortic shadow, but there is no absolute certainty of this happening

vary between *2.5 and 3 0 cm., the most common value (3.0 cm) is regarded as the* [illegible] are still not abnormal

[illegible] iterior

lowed

and its diameter measured at any optional point in an aged individual.

Conditions for measuring the diameter of the ascending aorta in the left anterior

... more favorable than in the right (Reich); with this ... Attempts ... 2) are often ...

distinguished in the lower ... e
pulmonary artery (Frik) unless the aorta is definitely ... y
... Much more regularly this posterior wall can be outlined at a ... of the pulmonary artery and nearly at the ... n

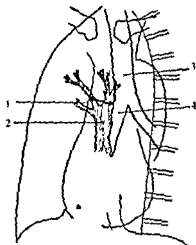


FIG 262 —Measurement of diameter of ascending aorta in the left anterior oblique position

1 Anterior boundary of ascending aorta
3. Trachea
4 Right bronchus

... with- stands ... can
be measured. With this ... rmal
build between the ages of 20 to 50 years are 2 5 to 3 0 cm, ... com-
mon figure, represents the upper limit and is exceeded by 3 to 5 mm only in individuals above average size, broad in build, and at an older age.

Measurements obtained in this way have the same limitations as those in the ... and confirmation by studying the ... oblique
positions are required as well ...
method.

The Kreuzfuchs method is based on the fact that the arch of the aorta after the left subclavian artery departs, near the isthmus, crosses and lies directly on the esophagus (fig. 264). This close anatomic relation is easy to recognize when the

esophagus is filled with barium. At the level of the aortic knob, through apposition of the aortic arch, the left border of the esophagus is indented and often bends locally to the right the "aortic bed" (Kreuzfuchs) (*fig. 265*). The *orthodiagraphic distance* from the outermost point of the knob to the deepest point of the aortic bed furnishes a good idea of the diameter of the aortic arch where it crosses the esophagus.

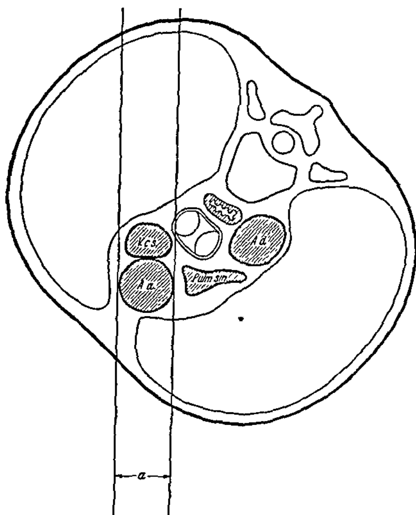
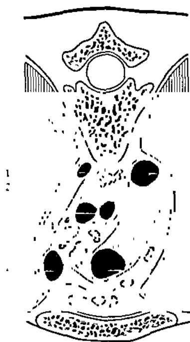


FIG 263 —Measurement of the ascending aorta in the left anterior oblique position The width of the vascular band (a) at the level of the bifurcation is determined exclusively by the ascending aorta

Naturally this prevails only when this section of the arch runs in the direction of the ray, that is, sagittally, only then does the projection of the aorta correspond to its circular cross section and is the measurement related to the diameter of the aortic lumen. This presumption is actually fulfilled in adults under normal conditions since only the proximal pretracheal part of the arch, the "*frontal section*" (v. Jagič and Kreuzfuchs), passes obliquely through the thorax, the distal section of the aorta,

coming under consideration here, assumes almost exactly a ventrodorsal course and consequently is called the "sagittal section" of the arch (fig. 264)

It is different when the aorta is elongated and displaced upward by elevation of the diaphragm Then, the distal section of the arch has an oblique rather than a sagittal course. Measurements obtained with sagittal projection in these cases do not



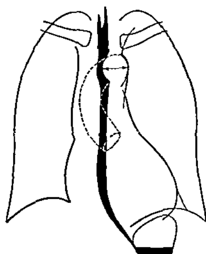


Fig 264 Fig 265

Fig 264 —Transverse section through the thorax at the level of the aortic arch (fifth thoracic vertebra) according to Pernkopf The aortic arch opened from above One recognizes the proximal oblique section of the aortic arch and the distal sagittal segment as well as the close positional relation of the latter to the esophagus

1 V azygos
2 Esophagus
3 Trachea
4 V anonyma dext
5 Aorta descendens
6 Aortic arch opened from above
7 Ascending aorta

Fig 265 —Measurement of the diameter of the aortic arch by Kreuzfuchs' method

represent the diameter of the aorta (fig 266a) because the point farthest to the left is not identical with the point on the left aortic border opposite to the site of esophageal crossing, rather it corresponds to a point more or less dorsad and laterad from that site Sagittal projection in these cases yields values which are distorted to different degrees depending upon the obliquity and the length of the section beyond the site of crossing.

Weiss and Lauda drew attention to a source of error in the Kreuzfuchs method which carries weight because the resultant variable is inconstant according to the degree of aortic obliquity Moreover, increased obliquity of the aorta is extremely

common. All elongations and dilatations of the thoracic aorta or elevations of the diaphragm can separate the limbs of the aortic loop laterally and produce greater obliquity of its arch. Also, young children and some adults, owing to special spatial conditions, have a relatively more oblique aorta than most adults (Kreuzfuchs). Moreover scoliosis, convex to the left (H. Rosler) and shrinking processes in the right lung and pleura (Kreuzfuchs) often make the arch more oblique.

This greater obliquity of the distal section is recognized by the absence of the aortic knob since a knob results only if the distal section of the arch runs practically in the path of the central ray. With upward pressure as well as with elongation and dilatation of the aorta, one sees in the posteroanterior position, in place of a knob, the

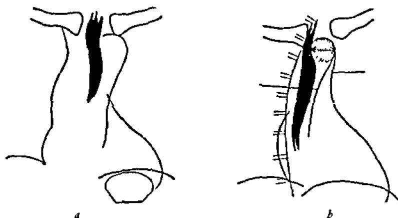


Fig 266.—Corrected Kreuzfuchs measurement Elongation of the aorta makes it impossible to perform Kreuzfuchs' measurement with dorsoventral projection (*a*) in a manner free from objection since the part of the aortic arch which the esophagus crosses, proceeds obliquely to the direction of the ray Only with slight rotation toward the right anterior oblique position (*b*) does the aortic knob appear On the appearance of the dark aortic knob one realizes that the part of the aorta crossing the esophagus proceeds in the path of the central ray Only then is the measurement free from objection

more or less exposed arch passing into the left convex curve into the descending aorta which descends paravertebrally on the left.

In these cases the absence of the aortic knob makes the Kreuzfuchs measurement impossible in the posteroanterior position This becomes possible, however, if one rotates the patient to the left to bring the distal section of the arch into the path of the ray (fig 266b) (Fleischner, Zdansky). One recognizes this position by the appearance of a distinct aortic knob.

In the Kreuzfuchs measurement the patient swallows a mouthful of barium and orthodiagraphically the systolic distance is determined from the outermost point on the aortic knob to the deepest point of the aortic bed. The esophagus should be as full of barium as possible and the paste should not be too thin, otherwise excessive values are obtained When the aortic knob is distinct in the anterior view, measurement can be made with posteroanterior position When the knob is absent in this position owing to obliquity of the distal aortic arch, the patient must be rotated to the left until the knob appears This point should not be exceeded

Kreuzfuchs' measure is made from the mediastinal pleura covering the aortic

arch to the lumen of the esophagus. To ascertain the real value one would have to subtract, from this figure, twice the mural thickness of the aorta and the thickness of the esophageal wall, the mediastinal pleura, the subpleural connective tissue, and the connective tissue separating the aorta and esophagus. The thickness of all these tissues with the esophagus filled scarcely exceeds 3 mm. so that it has been proposed to subtract 2 to 3 mm. from the value obtained. We use the uncorrected distance since the error created by including the tissues mentioned is rather constant and relatively small. Under special conditions, however, the total to be deducted from the measured distance may be much greater Often this can be convincingly demonstrated when aortic atheromatosis is present, the sickle-shaped or circular calcium shadow may lie as far as 2 mm. within the outer boundary of the aortic knob, this must be ascribed to thickening of the aortic wall In some patients decompensated and edematous by virtue of circulatory disease, striking differences for aortic diameter are secured with changes in the amount of edema. It seems likely that the appearance and disappearance of edema permeating the mediastinal and subpleural connective tissue plays a role. The Kreuzfuchs method as well as all methods for measuring aortic diameter suffer from these sources of error

In general, the error created by including these extraneous tissues may be disregarded for practical purposes

When correctly performed, Kreuzfuchs method has the advantage that its sources of error and the possibility of making mistakes are much less than with all other methods devised for this purpose. Moreover, the measurement can be made in all but a few cases. Among these exceptions are· space-occupying mediastinal processes in which the relation of the aorta to the esophagus is altered or cannot be determined, processes in the lungs or pleura involving thickening or retraction which pull the aorta away from the esophagus or preclude demarcation of the aortic knob against the left lung field, pure mitral lesions and some cardiac anomalies with a high, elongated and dilated pulmonary artery which thrusts the proximal aortic arch upward so that the distal section of the arch disappears behind the pulmonary artery (Zdansky).

These limitations also exist with all other methods advocated for determining the diameter or circumference of the aortic arch. Since the others are technically more difficult and, to some extent, introduce new sources of error, they do not, in our opinion, offer any advantage over the original method of Kreuzfuchs. They will receive only brief mention

De Abreu and Kreuzfuchs as well as Lian and Marchal recommended measuring the distance from the clear tracheal band to the outermost point of the aortic knob instead of from the barium filled esophagus De Abreu and Lins also attempted a geometric reconstruction of the aortic diameter, they drew two optional sectors of the aortic knob and dropped midperpendiculars from them, the distance from the point where these perpendiculars intersected to the surface of the knob was measured and considered the radius of the aortic cross-section Finally, Kreuzfuchs determined the aortic diameter by covering the knob with a transparent circle of known diameter, the circle exactly covering the knob has the same diameter as the aorta

At most, the Kreuzfuchs method measures the diameter at the isthmus which, as the narrowest part of the thoracic aorta, some believe is not a standard for the caliber of the rest of the vessel This

objection seemed valid since measurements made on the aortas of cadavers failed to show any constant relation between the width of the ascending aorta and of the isthmus (Reich) In contrast to this undeniable fact stands the constancy of Kreuzfuchs measurement in thousands of cases and its relation to measurements of the ascending aorta This relationship would be completely incomprehensible if the isthmus displayed the same unpredictable variations of its cross-section during life as in the cadaver The contradiction between anatomic and roentgenologic measurements presumably depends upon a difference in postmortem contraction at the isthmus and in neighboring parts of the aorta and suggests an inconstancy of their relations which is absent during life

Undoubtedly very constant dimensional relations exist between the cross-section of the isthmus and the rest of the thoracic aorta during life This fact is reflected in the remarkable regularity of "aortic width difference" (Lenk), that is, the difference between the roentgenologic diameter of the ascending aorta and the Kreuzfuchs measure. According to Lenk this difference amounts to only 5 to 10 mm. and in our experience to 3 to 7 mm. Only through this constant relation does the Kreuzfuchs value obtain its full significance and permit one to estimate the diameter of the ascending aorta on the basis of the Kreuzfuchs method when the size of this section of the vessel cannot be determined exactly. If, for example, examination in the left anterior oblique position reveals that the thoracic aorta is equally wide throughout, the Kreuzfuchs method provides reliable information on the absolute width, that is, how much aortic dilatation may be present. An aorta which seems dilated by upward pressure can be shown to be completely normal while one seemingly normal in width, under some circumstances, can be recognized as dilated.

By adhering to the technic described and by excluding cases in which the Kreuzfuchs measurement is technically unreliable or impossible, values between 2 0 and 2.5 cm are obtained in healthy adults of 20 to 50 years At first, this breadth of variation may seem extraordinarily large. Actually, it is astonishingly small because an overwhelming majority of all normal adults have a standard value, so to speak, of 2 5 cm which also represents the upper limit Values of 2.7 to 2 8 cm may occasionally still be regarded as normal only in individuals above average in size and of powerful build while they indicate aortic dilatation in subjects of average size Values below 2.5 cm. are relatively common in individuals somewhat below average size and of delicate build and occasionally are found even in obese subjects, especially in women. In them a delicate vascular system corresponds to body build and therefore the figure by itself should not be considered abnormal. Strikingly low values are often seen in asthenic individuals and, in young people who are growing rapidly (p. 372), these must be interpreted differently

We lack personal experience on a large series of aortic diameters in children In those from 10 to 16, our values were between 1 6 and 2 0 cm Kreuzfuchs reported as normal values for children 5 years, 10 mm ; 9 to 10 years, 13 mm , 12 years, 14 mm., 14 to 15 years, 15 to 16 mm

From the end of the growth period until about the age of 50, the aorta tenaciously maintains its diameter in the opinion of Zdansky This holds providing atheromatosis, syphilis or hypertension does not lead to dilatation For adults of normal size, of normal build, and completely normal from the standpoint of circulation, the diameter of the aortic arch, as already mentioned, is 2.5 cm with only slight deviations above or below this figure; these variations scarcely exceed 2 mm This does not deny that the average diameter of the aorta continuously increases with age (Suter) but this

may be ascribed to arterial hypertension, atheromatosis, or syphilitic aortitis which raise the average value

At advanced ages, perhaps after the sixth decennium, values of 2.7 to 3.0 cm, reflect age deterioration of the aorta and the frequent physiologic increase of blood pressure, by themselves they should not be regarded as pathologic.

In this connection studies of the cadaver aorta are of interest Suter showed that the average circumference of the cadaver aorta increased continually with advancing age but that stretching tests on strips of aorta made it probable that this increase was merely a postmortem phenomenon and that, on the contrary, aortic circumference in vivo could be quite independent of age The delicate youthful aorta could be stretched more by a load than an old thick-walled one By the same token, it was probable that youthful aortas contracted more decidedly post mortem than older ones At all events one must consider that post mortem contraction (Gerlach, Hvilivitzkaja, MacWilliam Reuterwall) may alter aortic circumference to a varying extent according to its anatomic status and the time at which the necropsy is performed Consequently, findings in cadavers can be transferred only with caution and with certain limitations to the situation in vivo The great constancy of the aortic diameter, as determined roentgenologically, seems to support Suter's opinion

III. The Density of the Aortic Shadow

The density of the aortic shadow depends upon 1) the plane of projection, 2) the width of the vessel, and 3) its anatomic condition It is easy to understand that the aortic shadow is denser when the vessel proceeds in the direction of the rays than when it courses transversely or obliquely to them It is also obvious that the diameter of the blood column in the aortic lumen determines the density of the shadow In contrast, the thickness of the wall is less important. The deposition of calcium salts in the wall naturally results in a denser shadow. Visible clumps of mural calcifications are recognized as dense streaks which are implanted in the aortic shadow about 1 to 3 mm within its outer border (figs 269 and 275)

It is not only calcium in the form of large deposits or connected placques which intensifies the aortic shadow, microscopic mural calcification can increase the density of the aorta providing the amount is sufficient

The visible impression of the density of the aorta, moreover, depends not only upon the intensity of its shadow but also upon the contrast with the surrounding tissue Abnormally increased or diminished radiolucency of the latter can reduce the contrast, in the first instance, the aorta is effaced by the clarity of its vicinity and in the second the tissues in the environment have approximately the same density as the aorta

On the other side the loss of mediastinal fat and connective tissue in senility and cachexia seems to improve the contrast since less secondary radiation occurs in the substance-poor area around the aorta than under normal conditions This might explain the remarkably good definition of the aorta in senile, emaciated individuals.

Otherwise the density of the aortic shadow has limited diagnostic significance because of the lack of an objective measure for its evaluation *As a rule of thumb the* aortic knob should not be darker than the cardiac shadow of normal size, increased density of the aortic shadow may be assumed when the vessel can be followed through the clear tracheal band in the left anterior oblique position

IV. Aortic Pulsations

In discussing pulsations of the mediastinal shadow, those of the aorta were also mentioned. It was noted that aortic pulsations may be considered essentially an expression of rhythmic variations of intra-aortic pressure. Actually, the size of the excursions varies with blood pressure amplitude. This is most striking in aortic valve regurgitation where brisk, lateral systolic and slower, median diastolic pulsations are observed as the roentgenologic correlate of pulsus celer. Reversely, aortic pulsations are often scarcely perceptible with a small blood pressure amplitude of the rapid and poorly filled heart

Nevertheless, there is no strict parallelism between blood pressure amplitude and the size of visible aortic pulsations, since the latter are decidedly influenced by the anatomic status of the aorta, its tonus, and its rhythmic shift of position.

Reduced distensibility of the aorta from sclerosis or syphilitic scars reduces its pulsations (Fetzer) so that even a heightened pulse pressure amplitude from aortic valve regurgitation occasionally no longer provokes visible pulsations. With a compensated pure aortic insufficiency the absence of visible pulsations can be legitimately considered a probable sign of severe anatomic alterations of the aorta To be sure, powerful pulsations do not preclude severe mural changes since the aorta may remain sufficiently pliable when adequately preserved and distensible zones survive between severely damaged sections.

Moreover, early aortic injuries are even the occasion for augemented pulsations which may be ascribed to lessened solidity of the wall. Hubert and Fetzer observed such pulsations of the ascending aorta in the initial stage of aortic lues and Erdélyi in early atheromatosis.

The significance of tonic orientation of the muscular elements in the aortic wall has found too little consideration in respect to aortic pulsations. Tonus essentially influences the average width of the aortic lumen (p 371) and its ability to expand with increase of systolic pressure (Zdansky). As the tonus of the muscle fibers decreases, the aorta progressively distends with a systolic rise of blood pressure, with higher tonus, distension is less. As a matter of fact, often where a reduction of vascular tonus might be expected on good grounds, pulsations are strikingly large. This happens, for example, in aortic valve insufficiency, in infectious diseases and in severe *thyrotoxicosis The remarkably small pulsations in some patients, in many* with nephritis for example, is presumably connected with increased tonus of the muscle Tonic influences on vascular muscle fiber certainly affect the ability of the aorta to dilate systolically and precludes a definite inference about the anatomic status of the vessel wall from the size of visible pulsations

Finally roentgenologic pulsations are influenced by rhythmic shift of the aorta (Dietlen, Bickenbach, Zdansky and Ellinger). These are produced in the region of the ascending aorta by rhythmic changes in filling and in the position of the left ventricle and in the region of the arch and descending aorta from the systolic impact of blood ejected from the heart (Weltz). The systolic shift of the arch can be easily recognized by filling the esophagus with barium and by kymographic registration (Zdansky and Ellinger). Systolic shift of the arch is alleged to have much greater significance on the occurrence of pulsations of the aortic knob than pulsatory

changes in aortic width (Weltz), normally, the latter are said to be scarcely perceptible. Our roentgenologic studies do not substantiate this statement (Zdansky and Ellinger), *rather pulsations of the aortic knob are composed of measurable* changes in the width and position of the arch.

Apart from pulse pressure amplitude, the extent of systolic shift and stretching of the aortic arch depends upon the grade of luminal curvature, the amount of dislodgement, the status of the wall, and the width of the lumen. Increased aortic curvature from diaphragmatic elevation promotes its displacement and augments pulsatory positional changes (Weltz) Fixation by mediastinal scars may restrict this shift

Sclerotic rigidity of the wall favors pulsatory shift of the aorta (Vaquez and Bordet, Dietlen) and the movement may be jerky (Erdélyi)

V. Dynamic Dilatation of the Aorta

Apart from its anatomic status the width of the aortic lumen is determined by several functional factors among which are 1) the amount of blood ejected into the aorta, 2) the height of intra-aortic blood pressure, 3) elasticity of the aorta, and 4) the tonus of its muscle fibers.

The two factors mentioned first tend to dilate and the last two to narrow the vessel The diameter of the tube increases with the stroke volume and the blood pressure The elasticity and muscular tonus, on one side, oppose dilatation and, on the other, guarantee adaptation of the lumen to diminished filling or falling blood pressure These facts find further discussion on page 372 The resistance which the *aorta, functionally and anatomically normal, opposes to internal pressure is great* so that dilatation from increasing pressure is only slight Nevertheless, it may enlarge the aorta definitely (White, Sheldon, Bayley, Purks, Zdansky) (see table 6) but this disappears when pressure falls. Tschilow and Christoff observed aortic diameter to increase 4 mm. during a paroxysmal rise of pressure from 140 to 220 mm. Hg

TABLE 6.

		Blood pressure mm Hg	Aortic diameter Kreuzfuchs method
N R Subacute nephritis	4 22/1936	175/95	3 2 cm
	5/29/1936	110/70	2 7 cm.
R E Acute nephritis	5/6/1935	170/75	2 7 cm
	5/16/1935	155/60	2 4 cm *

* After weight loss of about 7 Kg

However, the ratio between the height of the blood pressure and the width of the aorta is not fixed since the latter depends upon the changing anatomic status of the vessel and, primarily, on the variable tonus of its muscle Muscular tonus seems to be a very effective opponent to aortic overdistention Its reduction may cause abnormal, marked dilatation even when the blood pressure is normal. This dynamic

dilatation, in a narrow sense, was known prior to roentgenology (Osler, Sahli, Fleckseder, Ortner). Its true frequency was appreciated, however, only when roentgenologic measurements were undertaken Thus, it is not rare in thyrotoxicosis, vascular neuroses, tabes dorsalis, and infectious diseases, especially those with endocarditis (Bayley, Purks, Allbutt, Zdansky), and it is occasionally seen in Addison's disease (Zdansky). When regulation of blood pressure is disturbed (Schellong, Strisower), dynamic aortic dilatation seems to be common (Zdansky)

Whether in individual cases vascular tonus is reduced by toxic or reflex factors or, as in rheumatic fever at times, by inflammatory aortitis (Chiari) are questions which concern clinicians and physiologists.

Sometimes dynamic dilatation first appears when examination is conducted in the horizontal position with optimal conditions for filling the heart and aorta, although the aorta is normal in width in the upright patient (Zdansky).

The initial section of the ascending aorta is affected very often and to a special degree. This happens particularly in aortic stenosis (Volhard) and less often in aortic regurgitation and hypertension even when there is no reason for suspecting reduced vascular tonus *This dynamic dilatation is produced by the impact of blood* ejected with great force (Volhard); when marked, the roentgenogram suggests syphilitic aortitis or fusiform aneurysm of the ascending aorta Under these circumstances the discovery of an aorta of normal width at necropsy may be surprising

With prolonged abnormal pressure and conditions of flow, the original functional dilatation tends to be replaced by anatomically fixed dilatation.

VI. The Hypoplastic Aorta (Aorta Angusta)

In clinical literature the congenital narrow aorta always played a great role as a predisposing factor in various disturbances and diseases. This conception was originally taken over from pathology (v. Rokitansky, Virchow), since it was based upon direct measurements of the aorta in cadavers, it seemed to be firmly established Soon, however, clinicians as well as pathologists doubted *whether these measurements* could be transferred to the situation prevailing in life without further consideration (Suter, Kaufmann, Jaffé and Sternberg, Holzmann, and others) It was correctly realized that postmortem contraction could greatly alter vessel width and, even if this were not the case, no positive conclusion on aortic width in life could be obtained since the height of blood pressure actively distending the vessel was unknown. Actually, stretching the cadaver aorta indicates that these vessels possess differing distensibility, and precisely the artery which is delicate and thin at death was much more distensible during life than a wider and thicker one. These facts show that the real width of the aorta cannot be determined in cadavers but only in living subjects and by roentgenology It also became evident that true hypoplasia of the aorta was much less common than many had assumed on the basis of pathologic, clinical, and even some roentgenologic reports. The incidence of aorta angusta seems to be overestimated even roentgenologically since many measurements neglected a basic presumption: as a rule the measurements were not made on recumbent patients but on erect ones. Consequently, true aortic width was often underestimated because

the aortic lumen narrows just as the heart becomes more or less smaller owing to orthostatic decrease in filling In individuals of normal build and entirely free from circulatory disease this narrowing is just as trifling as the reduction of cardiac size In thin, tall subjects and especially in ptotic, asthenic individuals, however, decreased cardiac filling and considerable aortic narrowing as well may erroneously suggest aortic hypoplasia since the complaints of the patient seem directly referable to it. Kreuzfuchs' measurement repeated in the horizontal position, however, shows in the vast majority of instances that as the volume of the well-filled heart now increases, the aortic diameter also becomes greater (fig. 45a) and attains, or at least approximates, standard size (Zdansky), in other words, hypoplasia of the aorta vanishes Reduction of the diameter of orthostatic origin can amount to 3 to 4 mm

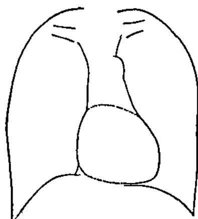


FIG 267 —Hypoplastic, short aorta Cor pendulum Asthenic subject, 22 years old Diameter of aortic arch by Kreuzfuchs' method while standing and lying = 1 8 cm

These facts indicate that one can assume the presence of aorta angusta only when the diameter is also measured in the recumbent patient by the Kreuzfuchs method (Zdansky)

The stipulation of limits below which one is justified in assuming narrowing of the aorta naturally remains rather arbitrary, obviously this also holds for the anatomic limits of aortic circumference (Beneke and others)

We assume that the aorta is hypoplastic when the Kreuzfuchs value is 2 2 cm or less in an adult of average size in the horizontal position and when no other reason (peripheral vasomotor weakness, oligemia, valvular stenosis, pericardial effusion, acute cardiac failure) explains the narrowing

In small individuals of delicate build, especially women, values of 2.2 cm and even 2 0 cm do not justify the diagnosis of aorta angusta These people, by no means, necessarily have a degenerative-hypoplastic constitution, perhaps their cardiovascular systems, delicately developed, correspond to their slighter physiques.

Low values are very common in children and usually represent a transient retardation of aortic growth Repeatedly we observed how the aorta of such individuals attained normal dimensions in the course of years The rarity of narrow aorta in adults certainly should not be ascribed to the fact that youngsters with slender aortas are premature victims to the development of vascular degeneration,

rather aortic growth is transiently retarded and finally the vessel attains normal dimensions.

Moreover, in true hypoplasia, the aorta is often too short as well as too narrow. This is expressed by striking brevity of the vascular band (Moritz); then the summit of the aorta is too far from the left clavicle (fig. 267) although the cardiac shadow is located relatively high. Despite cardiac elevation the central tendon of the diaphragm is fixed so that it descends sharply on all sides and the domes are flattened In other cases the elevated heart loses some of its support on the central tendon and is pendulous

Sometimes a narrow aorta results from unequal division of the truncus. This type is associated with an abnormally wide pulmonary artery whose arc protrudes into the cardiac waist (Laubry, Routier and Heim de Balsac)

VII. Diffuse Dilatation of the Thoracic Aorta

Diffuse aortic dilatation is always associated with some elongation of the vessel so that the ascending section bulges to the right anteriorly, the descending more to the left posteriorly, the summit is higher while the arch proceeds more obliquely than in the normal aorta.

The ascending aorta, bulging markedly to the right and forward, shifts over the superior vena cava and can overlap it so thoroughly that in the anterior view (fig 268a) the aorta forms the entire right border of the vascular band Finally, the powerful convex curve of the ascending aorta can project farther into the lung field than the right cardiac border Then, the latter usually seems shortened at the cost of the upper arc which rises abnormally low from the cardiac shadow. Normally the upper and lower right arcs of the mediastinal shadow are equal in length but now the upper arc is longer than the lower Above the ascending aorta, the pale shadow of the right innominate vein, concave to the right, appears and it can be followed to the clavicle

The elevation of the aortic crown permits the arch to appear on the left and its upper border may reach and occasionally even pass beyond the clavicle. The aortic knob is absent in most cases since the distal arch no longer has a sagittal course but runs rather obliquely. Instead of the knob, the distal arch passes into the descending aorta which protrudes to the left so that it proceeds somewhat paravertebrally on the left. Consequently, it is more exposed in the cardiac waist and runs convexly to the left; usually it can be followed in the cardiac shadow and often to the diaphragm.

As a rule, the heart is transverse since aortic elongation is invariably associated with dilatation, a lever effect rotates the heart around its sagittal axis (G. Schwarz) This transverse position causes greater projection of the left ventricular arc creating the impression of an aortic heart (p. 97). The angle of cardiac inclination is distinctly reduced.

The cardiac shadow may be normal or enlarged. Normal size of the heart without signs of left ventricular hypertrophy but with aortic dilatation speaks strongly in favor of aortic lues. Only when it appears in an individual above the age of 60, may pure atheromatosis be responsible.

In most patients with *aortic dilatation*, however, the cardiac shadow is somewhat enlarged. It shows an aortic configuration and signs of left ventricular hypertrophy and dilatation. The alteration of the heart, as a rule, is the result of hypertension or aortic regurgitation.

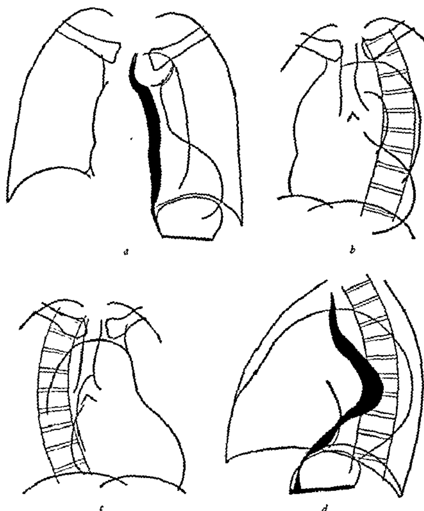


FIG 268.—Diffuse dilatation and elongation of aorta Female, 50 years old, with hypertension. The loop of the thoracic aorta bulges in massive arcs to the right anteriorly and the left posteriorly. The esophagus follows the proximal part of the descending aorta and bends with it to the left posteriorly. In the arch, mural calcification was evident (*a*) Anterior view, (*b*) left anterior oblique view, (*c*) right anterior oblique view, (*d*) left lateral view

The left anterior oblique position (fig. 268b) reveals a very characteristic picture of the dilated aorta The aortic loop passes through the thorax in a wide arc. The anterior border of the ascending aorta originates definitely lower from the cardiac shadow and bulges convexly toward the right lung field The distal part of the arch and descending aorta is projected into the vertebral shadow or, surpassing it, curves

into the left lung field While the normal aortic loop in this position is shaped like a *horse shoe*, with diffuse dilatation, the aorta forms nearly a sector of a circle or broad oval If conditions are otherwise favorable, *the outer as well as the inner edge* of the dense, markedly expanded aortic shadow can be recognized practically throughout and often is demonstrable within the clear tracheal band facilitating measurement at any optional place.

In the right anterior oblique position (fig. 268c) with a rotation of about 60 degrees, the transition of the widened vascular band into the *aortic arch is usually distinct, the latter bends into the descending aorta through the clear trachea* The anterior boundary of the descending aorta shows more definite contrast within the *retrocardiac field* in deep inspiration owing to its great density and the left-concave curve is more marked than normal.

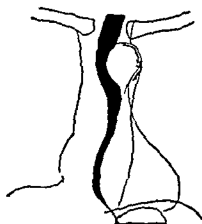


Fig 269—*Displacement of esophagus by a diffusely dilated and elongated aorta.* At the level of the aortic arch, the esophagus bends to the right Below, it follows somewhat the lower border of the arch and then in the retrocardiac section proceeds along the right border of the descending aorta and only just above the diaphragm it bends over the anterior wall of the descending aorta to the left of the hiatus oesophageus.

The alterations of the esophagus from dilatation of the aorta are noteworthy. The indentation which the barium filled esophagus experiences from contact with the aortic arch is deepened or the esophagus is displaced to form a large arc directed to the right-posteriorly Very frequently the esophagus follows the aorta for some distance below the lower limit of the arch so that it lies to the left of the midline; in this way the esophagus may show a diverticulum-like distension (Fleischner) in the absence of adhesions, this is supported by the same observation in aortic atheromatosis (figs. 268a and 269). Further on, the esophagus bulging to the left, often follows the descending aorta to which it may cling (Fleischner) and bend somewhat dorsad (fig. 268d). Behind the heart (perhaps at the level of D 7 to D 10) it often shows a bayonet-like kink to the left and forward (Oppler and Sielmann) to change to the opposite side over the anterior wall of the dilated aorta (fig. 270a and b). In this way the advance may go so far that above the diaphragm it must bend to the right to reach the hiatus oesophageus (v Falkenhausen, Fleischner). This displacement also

does not support the diagnosis of luetic aortitis as v. Falkenhausen believed, for it is found in aortic atheromatosis as well. The displacement is promoted by cardiac enlargement, particularly of the left ventricle when the esophagus is thrust between the heart and the dilated aorta and is forced to deviate (Zdansky). Sometimes the

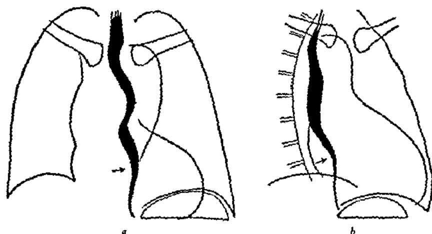


FIG 270.—Course of the esophagus with a diffuse dilatation and elongation of the aorta Hypertensive patient, 50 years old (*a*) Anterior view. (*b*) right anterior oblique view At first the esophagus follows the descending aorta which bulges to the left posteriorly and then in the retrocardiac division bends to the left anteriorly (arrow) to cross over the anterior wall of the descending aorta on its left border



FIG. 271 —*Alternating displacement of retrocardiac section of the esophagus to left and right* form a diffusely dilated aorta and hypertrophy and dilation of the left ventricle Male, 47 years old, with malignant nephrosclerosis

esophagus is compressed at this place, slowing or actually delaying the passage of barium, occasionally this may even lead to dysphagia (Hinteregger) At other times the esophagus escapes compression when it runs on the right side of the descending aorta (fig 269) In this way it may lie to the right of the midline and must approach the hiatus by an almost 90 degree kink to the left. Once we observed these forms alternate during an examination (fig. 271), this indicates that adherence of the aorta to the esophagus is not responsible. On the contrary, extensive mobility of the

esophagus within the loose mediastinal connective tissue is a prerequisite for such displacement.

These remarks should point out that diffuse aortic dilatation provides a well defined picture The extent of dilatation frequently can be measured directly in oblique positions especially when the vessel is abnormally opaque owing to marked dilatation or mural calcification. If, however, these situations do not prevail, Kreuzfuchs' method gives reliable information on aortic diameter. This measurement is unconditionally indicated when the diaphragm is elevated, for upward displacement of the aortic loop produces a picture which cannot be distinguished from diffuse dilatation without exact measurements (p. 357).

No definite decision about the cause (atheromatosis or syphilis) can be reached on the basis of the roentgen picture. This holds particularly when the cardiac shadow is somewhat enlarged owing to left ventricular hypertrophy and dilatation. Definite aortic dilatation in young and middle aged individuals should arouse the suspicion of lues. On the other hand, if the cardiac shadow is normal and signs of left ventricular hypertrophy and dilatation are absent, the probability of aortic syphilis is great if the patient has not reached the sixth decade; atheromatosis produces noteworthy aortic dilatation only at more advanced ages when arterial hypertension or aortic regurgitation coexist.

Calcium deposits, frequently visible as dots or linear shadows, indicate atheromatosis which commonly complicates syphilitic aortitis. These calcareous shadows often lie 2 to 3 mm inside the outer border of the aortic shadow (Figs 269 and 275) permitting one to demonstrate considerable thickening of the atheromatous aorta. At times in the left anterior oblique position, on films the deposits of calcium are interrupted at the orifices of the great brachiocephalic vessels but occasionally they also pass into the mouths of these vessels (fig. 275)

VIII. Elongation of the Thoracic Aorta

An aorta, diffusely elongated but of normal width or not essentially dilated is found especially in atheromatosis uncomplicated by existing or previous hypertension.

This elongation does not alter the vascular band in a striking manner if—as very often happens precisely in atheromatosis without hypertension—emphysema or general ptosis lowers the diaphragm Then, the elongated aorta has plenty of room in the long thorax. The length and slimness of the vascular band alone is noticeable

If the diaphragm stands in a normal position or when the elongation exceeds certain limits, the aortic summit rises and the loop experiences typical kinking The first kink is located in the middle of the ascending aorta and forms a blunt angle open to the left posteriorly, the second is located where the arch passes into the descending aorta; the third, in the descending aorta, consists of an outward retrocardiac bend, directed to the left posteriorly (*H. Rosler and White*). Often all three angles are visible even in the anterior view.

In the anterior view (fig 272a) the right border of the vascular band is elongated at the cost of the right atrial arc and projects as a rounded angle into the lung field

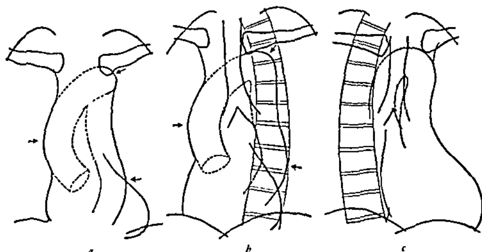


FIG. 272 —Elongation of aorta (a) Anterior view, (b) left anterior oblique view, (c) right anterior oblique view The arrows indicate the three typical kinks of the elongated aorta in the region of the ascending aorta, at the transition of the arch into the descending aorta, and in the retrocardiac part of the descending aorta

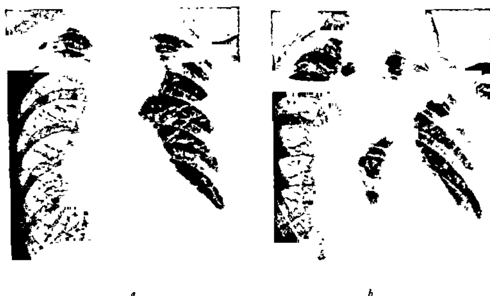


FIG 273 —"Low right position of aorta " (a) Anterior view, (b) right anterior oblique view

The aortic knob is high and protrudes markedly to the left (Fetzer), often it appears very dense even in the absence of aortic dilatation Frequently, the descending aorta bends convexly to the left even before entering the cardiac shadow, that is, in the region of the cardiac waist, or is demonstrable as a double contour, curved convexly to the left, within the cardiac shadow Sometimes the elongated descending aorta crosses the midline so that its right border appears as a convex shadow on the

right within or projecting beyond the heart (fig. 273a and b). The esophagus can be displaced in a different way (fig 274).

The kinks of the elongated aorta are surveyed best *in the left anterior oblique* position (fig. 272b). The first turns the vascular band as it emerges from the cardiac shadow toward the anterior chest wall, the second turns the distal section of the arch acutely in the vertebral shadow or even into the left lung field, the third makes the distal descending portion bulge convexly to the left within the vertebral shadow. Rarely, when the descending aorta crosses the midline to the right its shadow protrudes out of the spine into the cardiac shadow just below the bifurcation, then, its dark anterior boundary occasionally curves convexly to the right of the spine and within the cardiac shadow.

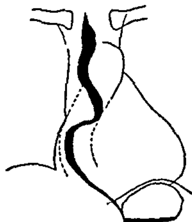


FIG. 274 —Marked elongation of dilated thoracic aorta with the descending aorta passing to the right Male, 65 years old, with hypertension (205/115 mm. Hg) and latent lues The esophagus crosses the anterior wall of the descending aorta at a place where this passes *beyond the spine* to the right and in this way attains the left side of the descending aorta. Then, it describes a second arc to the right, ventrad from the descending aorta, to bend toward the hiatus just above the diaphragm

In the right anterior oblique position (fig. 272c) the vascular band rises vertically and usually concavely to the left and then bends to the right into the aortic arch The descending aorta—more or less in an S-shape—is often visible in the retrocardiac field. If the descending aorta crosses the midline toward the right, its oval cross section may be seen in the retrocardiac space. In these cases the *barium-filled* esophagus draws over the anterior wall of the descending aorta to bend forward for a short distance (Pape)

The three kinks of the aorta are not always equally prominent, often one or the other is merely suggested In general, the amount of angulation is less marked if there is dilatation of the aorta. A greatly dilated aorta usually shows nearly equal rounding in its course despite considerable coexisting elongation

In the presence of three typical kinks, it is highly probable that all parts of the vessel are equally elongated and that the entire aorta is equally wide. Then, Kreuzfuchs' measurement with proper rotation to the left also gives reliable information concerning the diameter (p. 366).

IX. Sclerosis of the Aorta and Its Main Branches

Sclerosis without hypertension enlarges the roentgenologic diameter of the aorta insignificantly. Mural thickening as such cannot be distinguished from slight dilatation of the lumen and increases the roentgenologic measure but slightly. Elongation of the vessel stands in the foreground. The resultant roentgenologic alterations were described in the preceding section. Very frequently calcification of the wall (Kohler) is evident in sickle-shaped or circular densities in the arch More



FIG 275 —Lues and atheromatosis of aorta Female, 55 years old, with luetic aortic valve insufficiency Difference of blood pressure in the arms (right, 100/45 mm. Hg, left, 65/0 mm Hg) No pulse palpable in left carotid artery The extensive calcification of the aorta continues to the departure of the brachiocephalic vessels (intersection of arrows) Pulsus celer was not present in the aorta owing to the severe mural damage of the vessel (left anterior oblique view)

extensive calcification may be seen in the oblique position as linear shadows or scattered clumps Sometimes the orifices of the great brachiocephalic vessels are affected (fig. 275). Widespread calcification tends to occur when atheromatosis is combined with syphilitic aortitis Arteriosclerotic aneurysms of the thoracic aorta are rather rare (Ruffin, Castleman, and White), their symptomatology is identical with that of a syphilitic aneurysm

The density of the aortic shadow is not definitely changed by sclerosis per se (Baronowa), nevertheless small diffuse intimal calcium deposits may make the aorta somewhat denser For the most part, however, opacity of the sclerotic aorta seems greater owing to the sharper contrast against the surrounding structures (p 369)

The aortic knob may move markedly to the left, upward and forward The movement may amount to 1 cm. if the head is lifted during deglutition in sclerosis

(Erdélyi). This is explained as follows: During swallowing the rigid tube rises with the trachea and the left main bronchus more than a normal vessel which tends merely to flatten. An increased systolic shift of the entire aortic arch, directed craniad, may be seen in aortic sclerosis (Vaquez and Bordet, Dietlen). This shift may be jerky (Erdélyi); it may also be observed in early luetic aortitis (Hubert).

Sclerotic elongation of the aorta influences the cardiac shadow only in so far as the heart may be tilted and assume a transverse position. This causes greater prominence of the left cardiac border, now aortic configuration is more or less conspicuous (p. 207) since the marked protrusion of the aortic knob accentuates the



Fig. 276



Fig. 277

Fig. 276 —Atheromatous calcification of the abdominal aorta and splenic artery (The arrows point to the calcified splenic artery)

Fig 277 —Atheromatous calcification of the abdominal aorta (arrows) Left lateral view.

deepening of the cardiac waist. Left heart hypertrophy and dilatation are absent in pure aortic sclerosis.

Dilatation of the aorta, as already noted, is fairly marked only when arterial hypertension coexists; this is a frequent combination Usually all parts widen about equally and ordinarily the expansion is diffuse; more rarely the initial portion of the

[illegible] presence of

[illegible] may

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quently induces aortic sclerosis, however, one must count on its presence.

It is impossible to distinguish atheromatous from syphilitic dilatation of the aorta especially when left ventricular hypertrophy is absent. In hypertension and aortic regurgitation, aortic atheromatosis is usually combined with dilatation Only severe aortic dilatation or even moderate dilatation in the absence of signs of left

ventricular hypertrophy suggests the possible presence of syphilitic aortitis. While predominant dilatation of the ascending aorta speaks in favor of syphilis, it also occurs in atheromatosis; in fact the latter sometimes may provoke fusiform aneurysmal dilatation. Sometimes the aortic valves become insufficient so that pulsations like those of pulsus celer appear in the aorta. Apparently the atheromatosis can also cause aortic regurgitation

Atheromatosis of the abdominal aorta is frequently detected by its mural calcification If extensive it may be visible even on films with posteroanterior projection (fig. 276) but this is not common Much more often it is noted on films obtained in the lateral position (Assmann) as two parallel, continuous, or interrupted, dense linear shadows in front of the vertebral column (R Ledoux-Lebard, Calderon, and G. Ledoux-Lebard, Feldman) (fig 277) Atheromatosis of the abdominal aorta



FIG 278 —Aneurysm of splenic artery (case of J Haffner, Acta Radiol 17 602, 1936)

provokes aneurysmal widening far more often than in the thoracic section (Ruffin, Castleman, and White). The symptomatology is discussed on page 369

Calcification of large pelvic vessels is commonly noted in scout films of this region

More unusual are calcifications of the large mesenteric arteries (Grashey) and of the splenic artery (Assmann and Israelski). We have repeatedly observed a calcified splenic artery in old individuals. The double contoured, tortuous linear shadows at the level of the first lumbar vertebra extend toward the left upper abdominal quadrant in a very characteristic way and can hardly be confused with anything else (fig. 276). One young man also had generalized osteitis fibrosa (Assmann).

The roentgenogram of calcified aneurysm of the splenic artery has been described repeatedly (Lindboe, Haffner, Tixier, Baumgartner and Gardreaux, Natvig, Zdansky) (fig. 278) Large aneurysms are rather rare but nevertheless of great importance since they may cause fatal hemorrhage. Often they are asymptomatic although occasionally they are palpable as a pulsating tumor with a systolic thrill. Occasionally vague colicky pain is felt in the left upper abdomen. In some cases the solitary, walnut-sized shadow with a dense, at times discontinuous shell is located at

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deepening of the cardiac waist. Left heart hypertrophy and dilatation are absent in pure aortic sclerosis.

Dilatation of the aorta, as already noted, is fairly marked only when arterial hypertension coexists; this is a frequent combination Usually all parts widen about equally and ordinarily the expansion is diffuse; more rarely the initial portion of the ascending aorta is most affected. Dilatation alone does not establish the presence of sclerosis because even a decidedly dilated aorta of an old hypertensive patient may be free from sclerosis (Dormanns and Emminger). Since arterial hypertension frequently induces aortic sclerosis, however, one must count on its presence.

It is impossible to distinguish atheromatous from syphilitic dilatation of the aorta especially when left ventricular hypertrophy is absent In hypertension and aortic regurgitation, aortic atheromatosis is usually combined with dilatation Only severe aortic dilatation or even moderate dilatation in the absence of signs of left

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the level of the first lumbar vertebra Distinction from a calcified aneurysm of the left renal artery (see below) is possible only by pyelography.

Similar shadows occur in the equally uncommon calcified splenic cyst These cavernous structures, often interpreted as cystic lymphangioma (Lubarsch), can attain considerable size. One was as large as an apple (Gatersleben). The oval shadow was located just below the left diaphragm; it had a dense interrupted shell suggesting an echinococcus cyst. The lateral location, just beneath the left diaphragm, usually differentiates it from an aneurysm of the splenic artery. In respect to this position, one case reported as splenic artery aneurysm (G. Fuchs) may rather



Fig 279.—Aneurysm of renal artery The aneurysm projects into the upper half of the renal pelvis and compresses it a little (case of G Renck, Acta Radiol 7 306, 1926)

have been a splenic cyst. A splenic cyst confirmed by operation was described by Zdansky

Key and Åkerlund and Renck and Kment described the roentgenogram of calcified aneurysm of the renal artery (fig. 279). The oval or circular shadow, the size of a hazel or walnut, had the density of calcium. Its outer boundary is more sharply defined than the inner. Occasionally the circular shadow is briefly interrupted where the artery connects with the aneurysm In pyelograms the ring is located outside the renal pelvis and usually above the renal pedicle. This permits a definite distinction from a renal stone Gall stones are easily distinguished from an aneurysm of the right renal artery by a film obtained with sinistrodextral passage of the ray (Sgalitzer); gall stones are projected in front, renal artery aneurysm in the vertebral column

Renal artery aneurysm is rather rare and probably most of them are atheroma-

tous. However, one in a child, 8 years old, may have been caused by a mycotic injury of the vessel wall.

By compressing the outflow of the renal pelvis, the aneurysm may cause urinary stasis, colicky pain, and hemorrhage, rupture has been reported. In other cases aneurysm of the renal artery represents an asymptomatic incidental finding.

X. Syphilitic Aortitis

Although the beginning of syphilitic injury of the aorta is not detectable by x-ray and even severe alterations may be missed, roentgenologic examination has great diagnostic value because often it first reveals the existence of a silent diffuse aortic dilatation or aneurysm

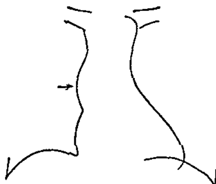


FIG 280 —Angular bend of the ascending aorta in aortic syphilis, the consequence of isolated dilatation of ascending aorta Male, 53 years old, with general paresis Diameter of ascending aorta = 3 5 cm Diameter of the arch = 2 3 cm

Neither diffuse or circumscribed aortic dilatation proves the presence of syphilis Diffuse dilatation is rather common in atheromatosis with hypertension or aortic valve disease as well as in older people If, however, left heart hypertrophy is absent and if the individual is under 50 years old, aortic dilatation, even if only amounting to 5 mm , is probably due to syphilis The probability grows with the extent of the dilatation and the youth of the patient

Of much greater weight for the presence of syphilis is circumscribed dilatation of the ascending aorta In the anterior view (fig 280) there is elongation, greater mid-right distance, and usually a blunt angular rounding of the right border of the vascular band (Eisler and Kreuzfuchs) As a rule the left anterior oblique position makes these signs more distinct Measurement reveals an increased diameter of the ascending aorta while the diameter of the arch may be normal or slightly increased The "breadth difference" (Lenk) is also increased above the normal of 3 to 7 mm

Isolated dilatation of the ascending aorta is not specific for lues since it also occurs in other conditions Thus, it is not uncommon in hypertension, especially in older patients, in aortic valve stenosis or regurgitation, in coarctation, that is, in situations associated with dynamic dilatation of the ascending aorta. Only when

these conditions are absent, may one consider a circumscribed dilatation of the ascending aorta as probably due to syphilis. On the other hand, the presence of left heart hypertrophy does not speak against syphilis since the latter frequently causes aortic regurgitation or hypertension.

Atheromatous calcification of the aorta in relatively young individuals is always suspicious of syphilis because aortic lues is often complicated by early atheromatosis

Increased pulsations of the ascending aorta (Hubert and Fetzer) do not seem to us characteristic of luetic aortitis.

XI. Aortic Aneurysm

While isolated dilatation of the ascending aorta tends to make the diagnosis of lues probable, the demonstration of a saccular or fusiform aneurysm almost ensures it because mycotic, traumatic, congenital, and erosion aneurysms comprise only about 10 per cent of all aneurysms of the thoracic aorta. However, the frequency of syphilitic aneurysms has sharply decreased in the last two decades while nonluetic aneurysms undoubtedly have become more common. In twenty-five years Zdansky encountered only two traumatic aneurysms of the descending aorta and saw only a few instances of mycotic aneurysm of the ascending aorta

The incidence of aortic aneurysms gradually decreases peripherally from the ascending portion of the aorta Often two or more aneurysms are found behind each other and they may merge directly Frequently daughter aneurysms rest on the larger sac

According to their shape, one can distinguish fusiform or even spherical and saccular aneurysms. In the first the aortic lumen expands symmetrically, the last is a protrusion whose broad or narrow base is located at one side of the lumen and occasionally the main stream and sac are connected only by a small orifice Daughter aneurysms are saccular and develop from rupture or special weakness of part of the wall Finally, dissecting aneurysm should be mentioned; through a partial rupture, the outer layers are undermined by blood entering under pressure to produce a crevice which may extend along the aorta for a long distance.

The roentgen findings of an aortic aneurysm are determined by its size, location, shape, and the status of its wall The course of the aorta makes it inevitable, however, for very similar shadows to be formed by diverse pathologic lesions developing in organs and tissues of the anterior and posterior mediastinum or their adjoining areas. Tumors of the lymph nodes and of the thymus, retrosternal goiter, tumors of the sympathetic chain, of mediastinal connective tissue, the pleura, pericardium, lungs, the thoracic bones, teratoma and dermoids, mediastinal and pericardial encapsulated effusions, tumors, diverticula and dilatation of the esophagus, abscess of the spine and aneurysms of the pulmonary artery must be taken into consideration in differential diagnosis. Saccular aneurysms in particular and, at times, pedunculated and mushroom-like aneurysms may closely resemble a tumor.

Kienbock and Lenk made important contributions to the recognition and differential diagnosis of aortic aneurysm; at present it is possible to distinguish an aneurysm from any of the lesions mentioned above For this purpose careful fluoroscopy

in different planes with contrast filling of the esophagus and films in definite positions after preliminary search are necessary. Sometimes, however, only angiocardiography or aortography permit a positive diagnosis (Dotter and Steinberg). Kymographic registration of the pulsations as a differential diagnostic measure does not contribute much to the diagnosis because aneurysms lined or filled with hard thrombotic masses do not pulsate and even tumors occasionally (p. 242) have or seem to display intrinsic pulsations (p. 391) Naturally angiocardiography may not reveal an aneurysm if it is filled with a clot so that the result is decisive only when the dubious structure is filled by the contrast material (Steinberg, Grishman and Sussman)

It is essential to ascertain the precise location of the questionable shadow and particularly to determine its relation to the aorta Usually this can be done by

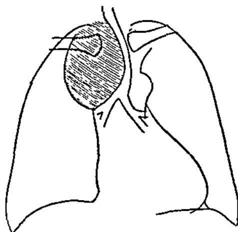


FIG 281 —Right sided retrosternal goiter with displacement of trachea and arch of aorta to left Female, 72 years old The displacement and compression of the trachea begins even in the cervical portion. The diameter of the aorta, displaced to the left, is normal, this is significant in the differentiation of a goiter shadow from the shadow of an aneurysm of the innominate artery Compare the great similarity to figure 292

fluoroscopy while the patient is rotated (Holzknecht) Then, one readily sees whether an aneurysm is actually present and the part of the aorta involved. If there [illegible]

other [illegible] an aneurysm [illegible]

[illegible] measured. Among a large number of luetic aneurysms scarcely one had an aorta of normal width (Kienbock and Thoma), at least the neighboring parts of the lumen were somewhat dilated Small fusiform or saccular luetic aneurysms as well as the rare mycotic, traumatic and erosion aneurysms and some saccular aneurysms of the sinus of Valsalva are exceptions to this rule. A luetic aneurysm is improbable if the diameter of the ascending aorta or of the arch is normal (fig 281) On the other hand, moderate aortic dilatation does not prove an aneurysm is present since dilata-

tion may accidentally coexist with some other mediastinal process. The age of the patient and the clinical manifestations should also be considered. If the patient is under 50 years of age and if the left ventricle is not hypertrophied, aortic dilatation speaks for aortitis and makes it probable that the dubious shadow is an aneurysm when other roentgen signs are compatible with this diagnosis.

The cardiac shadow need not suffer any change in size or shape owing to the aneurysm. Sometimes a small median heart (fig. 293) hangs like an appendage to a massive aneurysm (Groedel). Only aneurysms just above the valves tend to produce cardiac enlargement and an aortic configuration because they are often accompanied by aortic regurgitation. In other words, signs of aortic valve regurgitation favor the diagnosis of aneurysm in doubtful cases. This holds even when the questionable shadow is located far above the cardiac shadow or in the posterior mediastinum since the concurrence of an aortic regurgitation with a tumor or some other space-occupying mediastinal structure is less probable than a coexisting aneurysm. In some cases coincidental hypertension or a myocardial lesion makes the cardiac shadow large.

The shape of the otherwise normal heart can be altered by displacement Aneurysms of the ascending portion in particular as well as large ones of the arch can tilt the heart to produce a transverse position and cause greater bulging of the left border and aortic configuration.

In the differentiation from other mediastinal structures, the character of the questionable shadow has great importance. Above all, one notes· 1) shape and edges, 2) density and inner structure, 3) pulsations, if present; 4) growth, 5) resistance to radiation in certain cases.

In regard to the shape and edges, simple arching of the structure out of the mediastinal shadow is found most frequently with aneurysms while tumors and other space-occupying processes which protrude, are polycyclic or angular This rule is not completely valid Retrosternal goiter, tumors of the sympathetics, cysts and teratomas, encapsulated pleural or pericardial effusions, tumors receding from irradiation (lymphosarcoma, lymphogranuloma, lymphoepithelioma of the thymus) and other candidates for differential diagnosis may be ovoid, spherical, or bun-shaped and therefore produce simply arched or bun-shaped shadows like an aneurysm, on the other hand by unequal expansion (figs 287 and 290) or by daughter aneurysms (fig. 282), an aneurysm can display a polycyclic or nobby shadow. This also happens when several fusiform or spherical aneurysms are aligned behind each other and are projected into each other in different ways.

Usually the border of an aneurysm is smooth and shows, at most, small irregularities and indentations which extend toward the lung fields or the anterior chest wall, they are created by unequally thick cicatrices of the vessel and by adhesions to the surrounding structures (pleura, lung, chest wall). By infiltration, neoplasms like lymphoepithelioma of the thymus, carcinoma, and sarcoma, as well as lymphogranuloma, can show irregular borders, soft strands or a network of linear shadows can extend into the lungs All of these processes can, however, have a very sharp outline in the absence of infiltration. This is the rule in benign and relatively benign goiters, neurofibroma, fibroma, lipoma, teratoma, and dermoid cysts.

On the other hand the aneurysm may have a vague, faded outline when the sur-

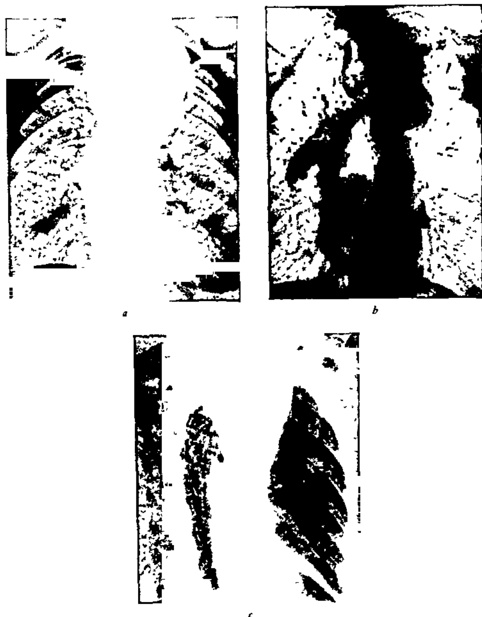


FIG 282 —Multiple aneurysms and daughter aneurysms of arch with extensive mural calcification. The fist-sized aneurysm of the aortic dome extends to the cupola of the left pleura (*a*) anterior view, (*b*) left anterior oblique view, (*c*) right anterior oblique view

rounding lung tissue gradually becomes permeated with blood as rupture impends (Assmann). In our experience the resultant shadow is homogeneous and broadly connected with the aneurysm (fig. 283).

Frequently bronchostenosis causes atelectasis and pneumonitis in the related sections of the lungs and consequently creates homogeneous, dense or soft, focal or

linear shadows, at times they become confluent and cannot be distinguished from *infiltrating new growths.* This happens most often in large arch aneurysms which have narrowed the left upper lobe bronchus (fig. 295).

The density of the aneurysmal shadow depends upon its size and its remoteness from the screen (Kienbock). The larger the aneurysm is and the nearer to the plane of projection, the more intense its shadow. Corresponding to its regular shape, in most cases the aneurysm is homogeneous or, at most, shows a light border If, however, the aneurysm is nodular or multiple aneurysms are projected into each other (figs. 282 and 290), according to the variable thicknesses of the parts penetrated,



Fig. 283 —*Aneurysm of the entire thoracic aorta with compression of the left main bronchus* and perforation of left lower lobe bronchus (necropsy) Perforation of the aneurysm was assumed as probable on the basis of nonhomogenous shadows in the base of the left lung Extensive mural calcification of aorta

different densities develop as with nodular tumors. Obviously a tumor or other mediastinal process can yield a homogeneous shadow when it has a regular shape

Calcification in the walls of many aneurysms produces a lack of homogeneity of differential diagnostic importance (figs. 282 and 283). These deposits result from the accompanying atheromatosis or mural thrombosis. Isolated small calcium deposits are often visible only on films while extensive calcification is readily seen on the screen. Calcium shadows are usually located 1 to 3 mm within the outer border of the soft tissue aneurysmal shadow and can often be followed for a long distance. The calcified shell of a retrosternal goiter, on the contrary, often shows nodular thickening and appendages extending into the soft tissue shadow. In other tumor-like structures, any calcifications present usually occur in the interior as irregular, foci, sometimes arranged in clusters, in teratoma they may show definite structure (teeth). Dermoid cysts may, however, have an extensive calcium shell (W. Muller).

Aneurysms often have *systolic expansile pulsations so that the shadow enlarges rhythmically;* these pulsations are extremely important in diagnosis. Their absence

in no way mitigates against the diagnosis, for only 50 per cent of aneurysms show intrinsic pulsations This might be anticipated since many aneurysms have hard or inelastic walls owing to scars or a thick lining of coagulum There need not be any pulsation even when the aneurysm has eroded the ribs or sternum to protrude from the anterior chest wall, this occurs when there is no systolic strain on the hard aneurysmal wall. When the communication between the sac and aorta is very narrow, when the cardiac stroke volume is small or the aneurysm is so large that even a normal cardiac stroke volume produces no noteworthy dilatation of the sac, visible intrinsic pulsations may be absent



FIG 284—Pulsating teratoma of the left anterior mediastinum, the size of a child's head, it had predominantly a hemangiomatous structure (necropsy) Male, 30 years old Systolic expansile, pulsating, homogenous structure broadly applied to the anterior chest wall and not demarcated from the aorta Left diaphragmatic paresis The normal width of the aorta permitted one to assume that no aneurysm was present despite the position and pulsation of the mass and suggested a tumor The tumor was removed by Prof Schonbauer Death two years later from local recurrence

Progressive increase of intrinsic pulsations speaks in favor of gradual thinning of the aneurysmal wall (Dietlen) especially when the sac enlarges, this holds if the stroke volume has not increased from improved cardiac strength or aortic regurgitation cannot be made responsible

The intrinsic pulsations can weaken with progressive thickening of the aneurysmal wall or by reduction of the stroke volume

Mediastinal structures which create shadows like aneurysms, as a rule do not show systolic expansile pulsations but rather pulsations transmitted from the heart or great vessels Usually a differentiation between transmitted and intrinsic pulsations is easy since the latter cause an enlargement universally in systole while the former are usually characterized by a systolic shift of the shadow in question. Differentiation may, however, be impossible when the excursions are very small or when systolic expansile pulsations are associated with transmitted ones, this is rather common with aneurysms located near a strongly pulsating heart or great vessel Moreover, it is not rare for large tumor masses around the aorta, especially

around the arch, to transmit pulsations simulating intrinsic pulsations. We have seen this repeatedly when tumors of the left upper mediastinum surrounded the aortic arch.

Moreover, vascular tumors (some sarcomas and hemangiomata) may show intrinsic pulsations since they enlarge systolically owing to the abundance of blood vessels. We have observed this in metastatic hypernephroma and once in a teratoma predominantly of hemangiomatous structure; its lively pulsations extended from the cardiac waist into the left lung field (fig. 284).

Aneurysms gradually enlarge although their growth as a rule is too slow for demonstration except when examinations are repeated over a period of months or years. Occasionally they suddenly increase in size and naturally this is ominous. In many cases, however, they remain stationary for a long time. Since all these possibilities can exist in mediastinal tumors meriting differential diagnostic consideration, *they lack decisive diagnostic significance. Nevertheless rapid growth speaks in favor* of malignancy in the widest sense of the word

Occasionally the fact that the shadow remains the same after radiation has diagnostic import, an aneurysm can be excluded without further consideration if radiation reduces the size of the shadow

The special symptomatology of aneurysms in different locations will now be discussed. The influence of neighboring structures is emphasized owing to their importance in differential diagnosis

1 Aneurysm of the Sinus of Valsalva

Saccular aneurysms of a sinus of Valsalva are rare; the right sinus is affected most often and only exceptionally is the left and posterior involved (Micks) They are not always syphilitic but they may develop on an arteriosclerotic basis or after endocarditis. In rare instances they are congenital. Congenital aneurysms presumably develop at the site of a congenital mural weakness and usually they are associated with other cardiac anomalies

Only isolated instances of sinus aneurysm have been described. After Kienbock properly interpreted a case in vivo as a sinus aneurysm, *Albrecht published the first* case confirmed by necropsy; his second patient did not come to autopsy

Aneurysmal dilatation of the right sinus produces a round shadow projecting to the right, forward, and upward which rests on the root of the aorta on a small base (Albrecht), it often shows intrinsic pulsations as well as mural calcification. Corresponding to its position the sternum and adjoining ribs may be eroded. The shadow resembles a mushroom, that is, it arises from the aorta on a small base. This is important in contrast to the more common aneurysm *low on the ascending aorta which* arises above the sinus to overlap the adjoining section of the aortic root Frequently these aneurysms also develop to the right and not rarely erode the sternum and ribs, very often they are associated with aortic valve regurgitation. In distinction from sinus aneurysms they usually have a broad base on the aorta

An aneurysm of the right sinus of Valsalva can rupture into the right ventricle and occasionally into the right atrium (Goehring, v. Hauser). In this way blood can *pass from the aorta into the right ventricle when the semilunar valves are closed* during diastole. The rupture of a congenital sinus aneurysm can occur in early life

but this event happens only in later years with an arteriosclerotic or luetic aneurysm. It may be associated with the sudden appearance of precordial distress and the development of a systolic-diastolic murmur and then dyspnea Cyanosis tends to be absent. The pulse pressure is usually increased owing to reduction of diastolic pressure. The heart enlarges moderately since the right ventricle dilates and hypertrophies owing to the influx of blood from the aorta

Aneurysm of the left and posterior sinus of Valsalva is even less common and will usually escape roentgenologic detection owing to its position, apparently it has not been described in roentgenologic literature An aneurysm of the left sinus could be forced to develop to the left and therefore could displace the pulmonary trunk to the left in front of it. Consequently compression of the pulmonary artery and cardiac enlargement might follow. Rupture of the aneurysm into the pulmonary artery, an occasional event, might lead to its dilatation and augmented pulsations (Scott, Zdansky). Usually the patient dies soon after rupture but if he survives the heart dilates under the influence of the arteriovenous shunt (p 134).

2 Aneurysm of the Ascending Aorta

Practically always aneurysm of the ascending aorta is syphilitic In very rare cases it may develop on the basis of a congenital weakness of the vessel wall, Taussig mentions two cases of this type, both of which were associated with an atrial septal defect Mycotic aneurysms of the ascending aorta seem to be very uncommon

The roentgen picture of an aneurysm of the ascending aorta differs in accordance with the position and shape of the circumscribed expansion of the vessel

Aneurysms at the base of the aorta sometimes extend to the sinus of Valsalva Roentgenologically, they cannot be positively differentiated from sinus aneurysms so that reserve is in order in assuming the presence of a sinus aneurysm in the absence of necropsy control

Aneurysm of the aortic root can completely evade roentgenologic demonstration, particularly if dorsally directed If directed ventrad, aneurysms at the root lie behind the pulmonary artery which may be displaced forward, compressed, or forced to protrude like an aneurysm of the left sinus of Valsalva and to simulate dilatation of the pulmonary artery trunk (Bordet and Lereboullet, Crawford and de Veer) This compression of the mainstem of the pulmonary artery may provoke considerable right ventricular dilatation These cases present a moderate or marked enlargement of the cardiac shadow, a mitral configuration, a protruding pulmonary arc, normal or reduced hilar shadows, and normal lung markings The actual aneurysm is not visible Nevertheless, consideration of the entire clinical picture often permits the correct diagnosis The absence of left atrial enlargement and of accentuated lung markings makes a mitral lesion improbable. The normal vascular shadows in the lungs are against cor pulmonale as seen in primary pulmonary sclerosis, they also make a patent ductus Botalli improbable. Clinical signs of pulmonary stenosis (loud, rough, systolic murmur over the pulmonic area transmitted to the lungs, cyanosis and dyspnea) in conjunction with the absence of any history suggesting a congenital anomaly raises the question of the possibility of a compression stenosis of the pulmonary artery This assumption is almost certainly

true if lues can be proven to exist or aortic valve regurgitation is present. In one case (Crawford and de Veer) a saccular aneurysm of this type impinged on the ventricular septum damaging the conduction system and causing heart block.

Occasionally an aortic aneurysm ruptures into the pulmonary artery (Weinberger, Zdansky); this leads to marked dilatation and massive systolic-expansile pulsations of the pulmonary artery and its intrapulmonic branches (*fig.* 285) One case of this type was followed for four years (Clerc, Bascourer and Froyet).

Small aneurysms of the middle division of the ascending aorta can completely elude detection if they are directed dorsad. If they emerge from the anterior aortic



Fig 285



Fig 286

Fig. 285.—Perforation of a supravalvular aneurysm of the aorta into the pulmonary artery (necropsy) Male, 55 years old. Clinically, aortic valve insufficiency with extreme dyspnea and cyanosis. Roentgenologically, marked protrusion of pulmonary arc with large pulsation of this and of the considerably enlarged hilar shadows Necropsy, two perforations, each the size of a lentil, leading from the aneurysm into the pulmonary artery

Fig 286—Large, knobby aneurysm of the ascending aorta which was fixed to the anterior chest wall and had led to a paresis of the left diaphragm Male, 43 years old

wall, occasionally even anterior views reveal them as dense nuclear shadows; in oblique and lateral views they protrude to project into the anterior mediastinum. Large aneurysms in this region usually produce striking changes in contour in anterior views since they pass beyond the left (fig. 286), or the right (fig. 287), or both cardiovascular borders (fig. 288a and b). In the first instance confusion is possible with a diffuse aneurysmal dilatation of the pulmonary artery, a (rare) mycotic or congenital aneurysm, or a persistent ductus arteriosus (p 442). With spherical or fusiform aneurysms, however, the vascular band does not ascend with parallel borders but widens biconvexly (fig 288b). Only when a saccular aneurysm is directed to the left and forward is differentiation from a dilated pulmonary artery difficult, the absence of roentgenologic and clinical signs of a congenital anomaly or a mitral lesion naturally permits one to assume dilatation of the aorta in these cases

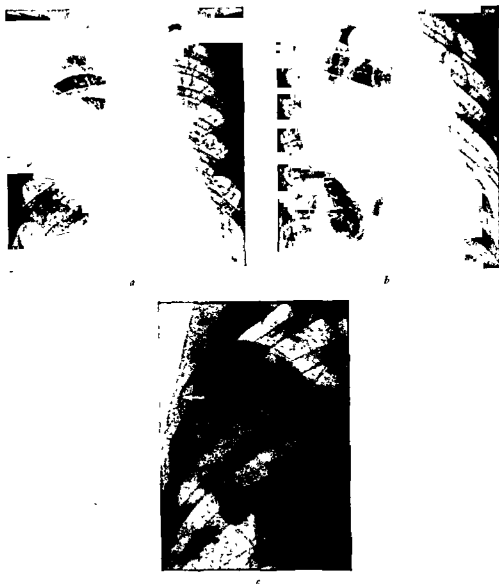


FIG 287 —Large saccular aneurysm of the ascending aorta extending to the anterior chest wall and eroding the sixth rib (arrow) (*a*) Anterior view, (*b*) right anterior oblique view, (*c*) rib erosion in the left anterior oblique view

Frequently an aneurysm of the ascending aorta directed forward erodes the sternum and ribs, early this can be detected only by x-ray (fig 287c) but views at suitable angles are necessary to demonstrate it

Aneurysms pointing to the right can extend nearly to the diaphragm and may overlap most of the right cardiac border (fig 289). Usually, however, they are higher and project beyond the cardiodiaphragmatic angle or into the right lung field. Occasionally they extend backward until they compress the right main bronchus.

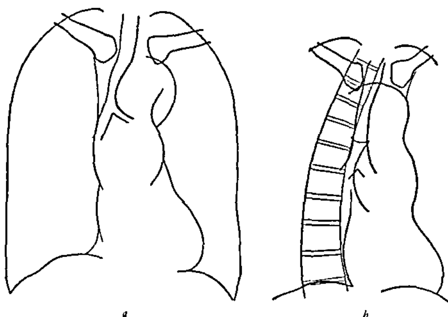


FIG. 288.—Double fusiform aneurysm of ascending aorta and arch. Male, 69 years old. (*a*) Anterior view, (*b*) right anterior oblique view. (Copy of films.)



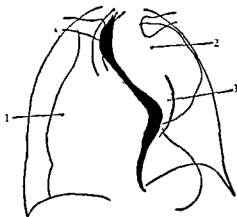


FIG. 289 FIG. 290

FIG. 289.—Large aneurysm of ascending aorta and arch. Aortic valve insufficiency. Male, 48 years old.

FIG. 290.—Large aneurysm of the ascending aorta and of the arch with dysphagia from compression of the esophagus. Aortic valve regurgitation. Male, 63 years old, who suffered from dysphagia for some months. At the level of the aortic arch, the esophagus bends to the right, below the bifurcation it bends to the left and posteriorly. Here, the esophagus was pressed flat between the two aneurysms so that the lumen was obstructed.

1. Ascending aorta
2. Arch
3. Ascending aorta

Often the resultant stenosis is announced by inspiratory wandering of the mediastinum to the right, sometimes it can be demonstrated directly on good films in the right anterior oblique position Extreme stenosis with widespread atelectasis is exceptional Fairly often the trachea and bifurcation are displaced to the left. Occasionally a large aneurysm of the ascending aorta extending backward displaces or even compresses the esophagus dorsad and to the left (fig. 290). Rather often with large aneurysms, the right leaf of the diaphragm is paretic (paradoxic respiratory shift of the elevated right leaf, inspiratory mediastinal wandering to the left) since

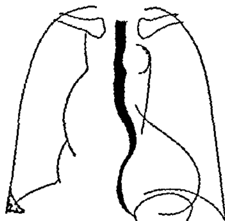


Fig 291

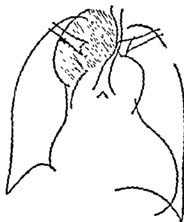


Fig. 292

Fig 291 —Predominant dilatation of the ascending aorta in syphilitic aortitis with aortic valve regurgitation Male, 63 years old Diameter of ascending aorta = 4 8 cm , diameter of arch = 3 5 cm Atheromatous calcification of arch. The small right pleural effusion is a common event in extreme widening of the ascending aorta

Fig 292 —Aneurysm of innominate artery (necropsy) Female, 50 years old Aortic valve regurgitation. Dullness below right clavicle Horner's sign on right Pulsating protrusion on right side of neck In the cervical area the trachea and esophagus were displaced to the left and compressed The sternal end of the right clavicle was destroyed, a common occurrence with innominate aneurysms The entire thoracic aorta was considerably dilated and showed extensive mural atheromatous calcification (Compare similarity to figure 281 of a retrosternal goiter)

the right phrenic nerve is damaged by stretching and adhesions as it skirts the aneurysm.

With marked dilatation of the ascending aorta, a persistent right pleural effusion frequently fills the costophrenic angle, probably as the result of stasis in the tributaries of the azygos (fig 291) Occasionally, such a hydrothorax vanishes without ascertainable reason after persisting for days or years Stasis from compression may widen the shadow of the superior vena cava in the angle between the aneurysm and right clavicle.

The size of the heart is not influenced by an aneurysm of the upper ascending aorta in the absence of aortic valve regurgitation. Nevertheless the heart, often tilted and transverse from aortic elongation, rests more broadly on the diaphragm and its transverse diameter is enlarged. The aortic arch is displaced to the left and up

(Lenk) so that the knob projects farther than dilatation of the arch, which usually coexists, would explain.

3. *Aneurysm of the Innominate Artery*

Luetic aneurysm of the innominate artery is always associated with aortitis and is usually combined with aneurysmal dilatation of the proximal section of the arch The convex, circumscribed shadow (Holzknecht, Assmann, Dietlen, Warfield) reaches from the right upper mediastinum into the soft tissue shadows of the neck (fig. 292). At the level of the shadow, the trachea may bend to the left or to the left and back or may be somewhat narrowed owing to compression. With forced breathing (Valsalva test) the trachea occasionally balloons indicating a local malacia (Sgalitzer). As a rule the barium-filled esophagus also shows a curved displacement in the lower neck and upper chest. The esophagus bends to the left at the level of the aneurysm and consequently turns to the right at the level of the arch. The second bend corresponds to the deepened aortic bed. Measurement of the aortic diameter by Kreuzfuchs' method usually shows that the arch is considerably dilated, a point of major diagnostic importance. Where the esophagus bends to the left, movements are occasionally transmitted from the systolic expansile pulsations of the aneurysm. The aneurysm may be lifted by swallowing or coughing to an extent about equal to the corresponding movements of the aortic knob At times a very typical erosion of the sternal end of the right clavicle (fig. 292) or of the adjoining part of the sternum is observed Moreover, a paralysis of the right diaphragm may follow injury of the right phrenic nerve. Sometimes the patient is hoarse owing to paralysis of the right recurrent laryngeal nerve.

An aneurysm of the innominate artery may closely resemble a retrosternal goiter or a glandular tumor or cyst of the right side of the mediastinum. Some similarity to a thick Lobus venae azygos has been noted (Warfield). Actually the distinction from a goiter or other tumor of the right upper mediastinum may be very difficult. Filling the esophagus with barium then may be very informative. The presence of an S-shaped course of the esophagus just mentioned plus the demonstration of considerable widening of the aortic arch are valuable signs in favor of aneurysm Goiters and other tumors displace the esophagus as well as the aorta to the left or to the left and down without affecting aortic diameter. A normal Kreuzfuchs' measurement speaks strongly against aneurysm. The pulsations transmitted to the esophagus are important since they indicate systolic expansile pulsation of the mass. Furthermore, an innominate artery aneurysm does not displace the aorta to the left and down like a retrosternal goiter does (Lenk); on the contrary the aortic knob remains high owing to marked dilatation of the arch. This rule has, however, exceptions. Finally an aneurysm is less probable when the movements of the dubious shadow produced by coughing and swallowing are completely independent from corresponding movements of the aortic arch.

4 *Aneurysms of the Arch*

These aneurysms are rich in symptoms because the arch is in close relation to many different organs.

Fusiform aneurysms of the proximal section of the arch usually make the vascular band club-shaped (fig 293) in the anterior view. This form is also characteristic in the right anterior oblique position In the left anterior oblique view the upper and lower borders are distinct if the aneurysm is sufficiently large so that it is easy to determine the diameter and the length of the dilatation directly.

Fusiform or spherical aneurysms at the vertex of the aorta and of the distal arch bulge to the left so that a round, large shadow projects into the left lung field in place of the aortic knob (fig. 294a and b) For these aneurysms as well, the left anterior oblique position is particularly informative since this position permits a survey of the entire length of the aorta and often allows direct measurement of its

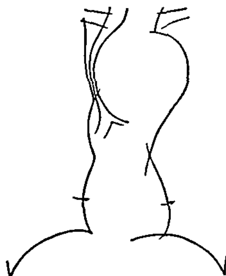


FIG 293 —Saccular aneurysm of arch with displacement and stenosis of trachea and left bronchus (necropsy) Male, 56 years old Inspiratory mediastinal wandering to left (arrow) as the result of left sided bronchial stenosis Small median placed heart

diameter The aortic window is narrowed from above whereby the lower convex edge of the pathologic shadow is contrasted sharply against the small clear band of the left main bronchus. This bronchus must pass around the lower edge of the aneurysm to reach the root of the lung, even on fluoroscopy one can see how the bronchus, elongated and displaced caudad, is considerably narrowed (see below)

The anterior view often shows distinctly how the trachea is also displaced, elongated, and compressed In fusiform aneurysms, the displacement is to the right or to the right and backward if the proximal section of the arch is involved, more rarely, displacement is to the right and forward when a saccular aneurysm moves between the trachea and spine (fig 298) Often the trachea forms the right border of the vascular shadow (fig 293) Tracheal compression may progress until only a cleft of the clear lumen remains With coughing or pressure (caution!) the lumen may widen distinctly indicating malacia (Sgalitzer). Moreover, the extent of compression and the amount of tracheomalacia is rather independent of the size of the aneurysm and the degree of tracheal displacement It seems that in relatively young individuals

the trachea is compressed and softened more often while the rigid airway of older individuals, supported by calcified rings of cartilage, offers stronger resistance to the pressure of large aneurysms Small saccular aneurysms shifting between the trachea and spine tend to produce severe stenosis presumably since the membranous posterior tracheal wall opposes less resistance.

Tracheal displacement by arch aneurysms may closely resemble the dislocation produced by a retrosternal goiter. Nevertheless a retrosternal goiter tends to displace even the cervical trachea (Kienbock) since a plug-shaped appendage of the goiter

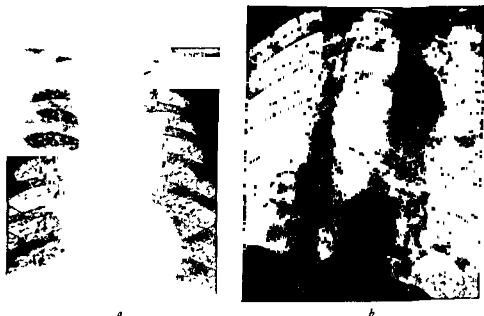


FIG 294 —Arch aneurysm Male, 42 years old, with left-sided paralysis of recurrent laryngeal nerve (*a*) Anterior view, (*b*) left anterior oblique view (The double feathered arrow indicates the dome of the arch aneurysm The two simple arrows designate the anterior and posterior sides of the moderately dilated descending aorta)

often extends into the neck, displacement from an arch aneurysm definitely affects only the intrathoracic trachea and tends to cause more circumscribed dislocation. The same situation also holds for the esophagus (Kienbock). Since dermoids are frequently located in the upper thorax, they behave like aneurysms in this respect

Another finding differentiating arch aneurysms from retrosternal tumors is the mobility of the shadow on coughing and swallowing. Under these circumstances the aneurysm rises no farther than the aortic arch while tumors may show much greater excursions. Tumors do not comply with this rule when they are adherent to the aorta or chest wall, on the other hand, rare pedunculated aneurysms may be fixed to the trachea (Schatzki).

The bifurcation is displaced to the right and down by large aneurysms and may arrive near the right border of the mediastinum (fig. 293). The left main bronchus is most endangered for the aortic arch passes over it. It makes a long detour around the right lower border of the aneurysm to reach the root of the lung and consequently suffers considerable elongation and more or less compression. At first this is betrayed

merely by inspiratory mediastinal wandering to the left, it may, however, lead to atelectasis and pulmonary collapse if compression becomes extreme Then, apart from mediastinal wandering the left lung field is cloudy and smaller, the intercostal spaces narrow, the mediastinum displaced to the left, the left diaphragm elevated and its movements reduced with respiration Large arch aneurysms can cause intense, homogenous darkening of the upper left lung field Below, this darkening tends to end rather abruptly and convexly It is produced by the aneurysm as well as the upper lobe which is compressed and atelectatic from the associated bronchial



Fig 295 —Aneurysm of the arch with atelectatic retraction of left upper lobe owing to stenosis of the left upper bronchus A second aneurysm emerges from the ascending aorta, its left border is indicated by a calcified strip curved to the left convexly. Male, 56 years old

stenosis (fig 295) In these cases differentiation from a tumor of the left upper lobe (Lenk), from a large mediastinal cyst or a neurofibroma of the sympathetics may be difficult or impossible Sometimes filling the esophagus with barium provides a diagnosis because the aortic bed is found normal, this argues against the presence of an aneurysm. With large aneurysms often focal and linear shadows are found at the bases of the lungs, usually inflammation provoked by retained secretions is responsible for them

Occasionally an arch aneurysm causes stenosis of the left pulmonary artery Then, the hilar shadow becomes small, the left lung field light and its markings sparse, the vessels emerging from the hilus look short and small (fig 296)

An aneurysm at the vertex of the aorta and of the distal section of the arch always forces the esophagus to bend to the right, the radius of the bend corresponding to the width of the aneurysm Since the esophagus is compressed, dysphagia may develop Esophageal displacement is not always purely lateral for it may bend simultaneously forward or backward depending upon the location of the aneurysm Some aneurysms force their way between the trachea and spine to press the trachea forward to the right (fig 297) and the esophagus to the right, others develop in the



FIG 296.—Aneurysm of the arch with compression of left pulmonary artery Owing to this compression the left hilar shadows are small and the vessel shadows of the left lung are sparse, small, and short. The entire lung field is remarkably light.



FIG 297.—Saccular aneurysm of the arch, apricot in size, which displaced the trachea to the right and forward and compressed it considerably (arrow) The esophagus was displaced exclusively to the right. Male, 43 years old, with extreme dyspnea and tracheal tug (Oliver-Cardarelli sign). (*a*) Anterior view, (*b*) left anterior oblique view (esophagus partly filled with barium)

space between the esophagus and spine and displace the esophagus to the right and forward (fig. 298); occasionally an arch aneurysm protrudes between the trachea and esophagus pressing the trachea forward to the right and the esophagus backward to the left (Gluck). In all these cases often the stenosis is rather marked and associated with dysphagia

Frequently large arch aneurysms cause left diaphragmatic paresis with its results owing to injury of the left phrenic nerve

The voice may become hoarse from paralysis of the left recurrent laryngeal nerve.

Pressure erosion of the spine is common (Haenisch) and gives rise to pain and paralysis (fig. 298). These erosions are scarcely demonstrable with fluoroscopy but



Fig. 298.—Erosion of some thoracic vertebra by an aneurysm of the arch (arrow) The barium-filled esophagus describes a large arc around the right anterior border of the aneurysm (left lateral view).

they can always be found on films taken in proper planes They are easily missed when films are obtained only in one direction Where the pulsating aneurysm contacts the surface of the corresponding vertebra, concave, circumscribed, but often deep, defects summate in an excavation, arcade in form, over a large or small section of the spine. The concave border of the defect in a single vertebra depends upon the greater resistance which the cartilaginous disc and the upper and lower borders of the vertebra offer in comparison to the lateral walls and anterior walls. The polycyclic borders distinguish pressure erosion from destruction by neoplastic processes Confusion with tuberculous spondylitis is improbable

5 Aneurysm of the Descending Aorta

Aneurysms of the descending aorta display different pictures according to their site in the upper and lower division Those of the proximal half bulge behind the heart toward the left lung to resemble a neurofibroma of the left sympathetic chain Fairly often these aneurysms, developing to the right, push the esophagus away from

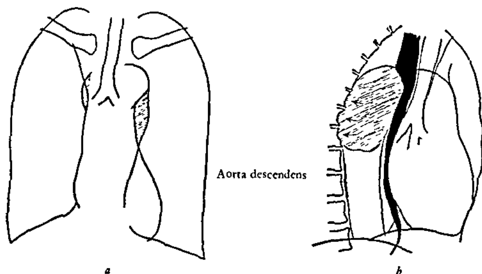


FIG 299 —Saccular aneurysm of the proximal part of the descending aorta with erosion of vertebra Male, 60 years old, who suffered from dysphagia for several months The aneurysm had developed between the esophagus and spine and compressed the first and eroded the second In this way it became evident on the right in the anterior view. (*a*) Anterior view, (*b*) right lateral view



FIG 300 —Double fusiform aneurysm of the descending aorta with compression of the esophagus The aneurysm is projected into the heart and is perceptible within the cardiac shadow as two humps. The esophagus is displaced to the left and anteriorly and compressed by the aneurysm so that obstruction developed Moreover, this patient had a large abdominal aneurysm One also sees an indentation of the pars cardiaca of the stomach from the right by the aneurysm

the spine and compress it to cause dysphagia (fig 299) Aneurysms of the distal retrocardiac descending aorta may be completely hidden by the heart (fig 300) unless they are sufficiently large to project beyond the left cardiac border (fig. 301a). The right border of the aneurysm is practically never delineated in the posteroanterior position since it is projected into the shadows of the heart and spine, its left border is frequently visible as a dense shadow, curved convexly to the left.

This shadow may simulate the supradiaphragmatic bend of an elongated aorta (p 379) or a cold abscess.

In one case (H Rosler), a large saccular aneurysm developed on the basis of a "low right position" (p 379) It projected to the right of the esophagus into the right hemithorax, the shadow, the size of a child's head, appeared to the right behind the heart The esophagus was displaced forward and to the left

As a rule aneurysms of the descending aorta can be surveyed best in the left anterior oblique position (fig 301b) In this position the dorsal aspect of the descending aorta can be followed as a simple arch within the vertebral shadow (p 46). *If*

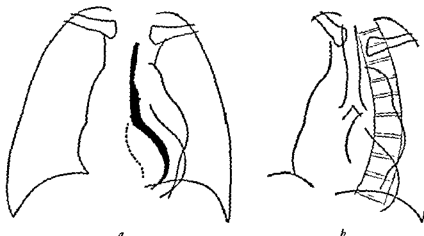


Fig 301 —Fusiform aneurysm of the descending aorta Aortic valve regurgitation Male, 32 years old, with general paresis (*a*) anterior view, (*b*) left anterior oblique view The right boundary of the aneurysm is not visible with dorsoventral passage of the ray (dotted line) The esophagus passes over the anterior border of the aneurysm to the left and bends toward the hiatus oesophageus to the right just above the diaphragm

the descending aorta has a fusiform or spherical dilatation, a corresponding protrusion rests on it *Naturally the right anterior boundary is perceptible only when the* aneurysm is very large since this edge is partly lost in the clear retrocardiac space and is partly superimposed by the cardiac shadow

Usually the esophagus is displaced to the right by aneurysms of the descending aorta Occasionally it is wedged between a retrocardiac aneurysm and the heart so that obstruction develops Sometimes it advances over the anterior wall of the aneurysm so far to the left that above the diaphragm it must bend to the right to reach the hiatus

Differentiation of a retrocardiac aneurysm from a cold abscess of the spine is often possible only with films of the spine with posteroanterior and lateral views. Whereas aneurysms often provoke the erosions mentioned on page 403 (fig 299b), a cold abscess is usually associated with signs of local tuberculosis In rare cases an aneurysm of the descending aorta erodes the ribs paravertebrally (Sacks)

Aneurysms of this section of the aorta can be distinguished from the supradiaphragmatic bend of an elongated aorta since the latter usually shows elongation

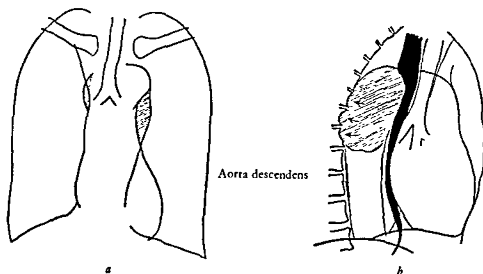


Fig 299 —Saccular aneurysm of the proximal part of the descending aorta with erosion of vertebra Male, 60 years old, who suffered from dysphagia for several months The aneurysm had developed between the esophagus and spine and compressed the first and eroded the second. In this way it became evident on the right in the anterior view (*a*) Anterior view, (*b*) right lateral view.



Fig 300 —Double fusiform aneurysm of the descending aorta with compression of the esophagus The aneurysm is projected into the heart and is perceptible within the cardiac shadow as two humps The esophagus is displaced to the left and anteriorly and compressed by the aneurysm so that obstruction developed Moreover, this patient had a large abdominal aneurysm One also sees an indentation of the pars cardiaca of the stomach from the right by the aneurysm.

the spine and compress it to cause dysphagia (fig 299). Aneurysms of the distal retrocardiac descending aorta may be completely hidden by the heart (fig 300) unless they are sufficiently large to project beyond the left cardiac border (fig 301a). The right border of the aneurysm is practically never delineated in the postero-anterior position since it is projected into the shadows of the heart and spine; its left border is frequently visible as a dense shadow, curved convexly to the left.

vertebra with its concavity directed toward the hemorrhage may appear (Kjellberg). Sometimes an aneurysm of the abdominal aorta extends into the thorax

7. *Dissecting Aneurysm*

Dissecting aneurysm is a blood-filled space within the aortic wall which developed by rupture of the inner layers of the vessel, the entering blood gradually undermines and separates the outer layers of the vessel The passage created may dissect around the aorta or merely on one side and often follows along the vessel for a considerable distance Often a return perforation creates a second junction with the aorta at the distal end of the space so that the dissecting aneurysm represents a by-pass of the aorta

Dissection usually occurs in aortas which have lost their solidity by atheromatosis or luetic mural damage. In most cases the blood pressure is elevated, or exertion preceded the catastrophe Sometimes, however, no anatomic lesion or hypertension is present, then, an abnormal susceptibility of the aortic wall, constitutionally conditioned, is held responsible for the dissection

Most often the dissection begins above the aortic valve Usually it proceeds transversely and consequently dissociates the valves to make them nonocclusive The second common site of initial rupture is the junction of the arch with the descending aorta

Dissecting aneurysms may be saccular or tubular In the first, a cavity containing blood projects above the surface as a hemispheric or oval mass In the second, a tubular formation accompanies the real aortic lumen for a long distance Very frequently the dissection extends from the supravalvular section of the aorta or arch to the abdominal aorta and even into the legs

Clinically the dissecting aneurysm usually starts dramatically with sudden pain which increases up to a sense of impending dissolution It is rare for the sudden pain to be absent ("silent dissecting aneurysm"). Then, collapse supervenes with pallor, fever, and leukocytosis Sometimes a diastolic murmur appears when a supravalvular tear renders the valves incompetent Through distortion, thrombosis, or occlusion of the orifices of large vessels, loss of consciousness, paraplegia, coldness, and loss of pulse are noted, depending upon the field supplied by the vessel Left pleural effusions are not unusual

In 80 per cent of the cases death occurs in a few days If, however, organization and endothelial relining of the wall of the dissection occur, a stationary situation develops and may be called healing This happens in about 10 per cent of the cases (Weiss, Kinney, and Maher)

This description suggests that the roentgen findings of dissecting aneurysm differ from those of true saccular or cylindrical aneurysm only in exceptional instances A difference in shading in the sense that dissecting aneurysm has a pale peripheral zone in contrast to the dense aortic lumen cannot be expected, the dissecting aneurysm is also filled with blood and must have the same density as the aortic shadow itself In one case Holzmann explained the lighter peripheral zone noted on the left side of the vascular band as follows a cross section of the dissecting aneurysm, as an accessory tube semicircular in shape, rested on the left border of the descending aorta, owing to its smaller diameter it was lighter than that of the main lumen of the aorta The roentgenograms, however, cast doubt upon this interpretation and suggest that the difference in shadows could have developed from the partial projection of the ascending and descending aorta with their aneurysmal dilatations into each other. The same reservations may be expressed about Canigiani's case. One patient of Kienböck and Weiss and three others (Kienböck) lack necropsy control. One dissecting aneurysm (Wood, Pendergrass, and Ostrum) confirmed by necropsy, had no roentgenologic signs which an ordinary aneurysm could not have produced. Even if this case and those cited above are accepted, the diagnosis is possible only by resort to the clinical picture and case history. With them roentgen examination is

of the ascending aorta and arch as well (p. 379). Furthermore, suitable films will show *whether an elongation* or *local dilatation* of the aorta is present.

6. Aneurysm of the Abdominal Aorta

Aneurysms of the abdominal aorta are much rarer than those of the thoracic aorta. While the latter are usually luetic, the former are ordinarily arteriosclerotic. Usually they are located where the coeliac or renal artery departs and lie on the left or, more rarely, on the right side, in front of the lowermost thoracic or upper lumbar vertebra They may be fusiform, spherical, or saccular. Larger aneurysms of the abdominal aorta, palpable as pulsating tumors, have a thrill and a systolic murmur. Frequently they are accompanied by severe intermittent crises or pain like that of renal colic and signs of peritoneal irritation. The crises are produced by pressure on the spinal nerves or sympathetic ganglia, the latter by traction on the peritoneum, inflammation and retroperitoneal bleeding Pain resembling angina abdominalis may be considered as vascular and visceral reflex events. Hemi- and paraparesis may result from erosion of the vertebra with compression of the spinal cord. Rarely the aneurysm mechanically compresses the stomach, intestine, or ureter and provokes functional disturbances in this way

Direct roentgenologic proof of an aneurysm of the abdominal aorta is obtained only when a homogeneous mass of shadows, not shifting with respiration, is contrasted against the gas-filled bowel, or when its wall is calcified, the latter event is common (Laubry, Farmer, Hollmann, Hartung, Kjellberg, Fuchs). Frequently an aneurysm can be visualized directly by the insufflation of air into the bowel or into the stomach (the latter by means of an effervescent powder) or the induction of pneumoperitoneum (Bottner, Laubry, Eskuchen, Nemenow, McClure, Farmer); against the clear background of intra- or extraintestinal gases the retroperitoneal shadow of an aneurysm projects toward the peritoneal cavity in strong contrast when the patient assumes an appropriate position Naturally echinococcus cysts, retroperitoneal hematoma and dermoid cysts as well may also produce round or oval shadows with calcified borders An unequivocal systolic expansile pulsation practically ensures the diagnosis of aneurysm (Laubry).

Aneurysm of the abdominal aorta may displace and compress the stomach and intestines. We have seen the stomach displaced markedly to the left and forward Compression of the Flex duodenojejunalis (Baird, Lester and Kirklin) and displacement of the gastrointestinal tract have been described (Kommerell and Roemheld). Naturally these displacements are not pathognomonic. Of much greater diagnostic significance are erosions of the vertebra, transverse processes and ribs (Eskuchen, Brailsford, Farmer, Nemenow, McClure, Hollmann, Baird, Lester and Kirklin, Kommerell and Roemheld). The twelfth thoracic and the first to third lumbar vertebra are affected most frequently; more rarely the left transverse processes of the upper lumbar vertebra are involved, destruction of the last two ribs is rarest. Films are required to demonstrate destruction of bone.

Rupture of the aneurysm into the retroperitoneal space may cause a *loss of outline* of the psoas muscle and kidney shadow on the corresponding side when the tissues become permeated with blood (Kjellberg); likewise a scoliosis of the lumbar

sure, normally persist but undergoes a partial regression and interruption The interruption occurs in the region of the right descending aortic root which establishes the connection between the fourth right branchial cleft artery and the descending aorta (fig. 303). By this interruption, the fourth left branchial cleft artery assumes the role of the surviving aortic arch while the right atrophies, so to speak, after the departure of the right innominate artery to the right subclavian artery.

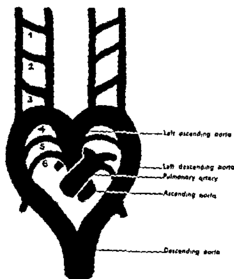


Fig. 302

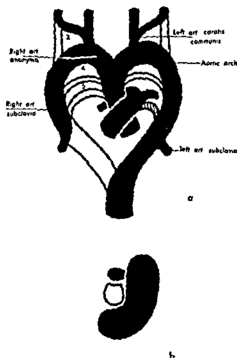


Fig. 303

Fig. 302.—System of the branchial arch arteries and the primitive ascending and descending aorta.

Fig. 303.—(*a*) Normal development of aorta and its large branches (*b*) Positional relation of normal aortic arch to the trachea and esophagus

1. Arcus Aortae Circumflexus Sinister

Occasionally the descending aorta proceeds caudad on the right rather than the left In this instance the aortic arch after crossing over the esophagus to the right in an almost horizontal direction enters the right hemithorax between the esophagus and spine, thereupon to become the descending aorta (Paul, Edwards, Grob, Franke) (fig 304). Embryologically, this anomaly is not completely explained Occasionally it is found with congenital anomalies of the heart and particularly with Fallot's malformation. In the roentgenogram (Roesler and White, Franke, Thurnher) one sees on the left a normal aortic knob and on the right the descending aorta as a pale, right-convexly curved shadow which projects dorsad and to the right of the superior vena cava (fig. 304). In the posteroanterior position below the normal aortic bed the

often decisive In some cases (Rosler, Gifford, and Bettes, Hoskin and Gardour) enlargement of the aneurysmal shadow in a few days indicated a dissecting aneurysm (see also Campbell, Jones and Langley)

The roentgen diagnosis of dissecting aneurysm is more probable when the real aortic lumen can be recognized within the aneurysmal shadow by virtue of calcium deposits, confusion with calcified thrombi lining the wall of an aneurysm must be avoided Roentgenologically demonstrable arteriosclerosis may occur in the newly formed canal (Weiss, Kinney, and Maher).

The diagnosis of a dissecting aneurysm can be established by angiocardiography when the condition of the patient permits (Robb, Golden and Weens, Dotter and Steinberg). Then, the wall of the aorta appears with a slender highlight between the lumen filled with opaque material and the equally opaque aneurysmal cavity. Naturally thrombosis in the aneurysm precludes this opacification and the irregularly defined opacified lumen of the vessel within the aneurysm appears as a denser shadow

Intramural hematoma must be distinguished from dissecting aneurysm In the former, blood permeates the aortic wall, locally or superficially after an intimal tear. A roentgenologic distinction from ordinary or dissecting aneurysm, if possible, would be exceptional

In one case (Hormuth) a shadow was found behind a fist-sized nonpulsating, purring protrusion to the right of the sternum, although resting broadly on the right side of the heart, it was sharply defined from the lung It was separated from the heart and aorta in oblique positions by a seam about 1 cm in width This chest wall hematoma (false aneurysm) followed perforation of an aneurysm of the ascending aorta According to the author, the erroneous assumption of an aneurysm might have been avoided if proper consideration had been given to the absence of rib erosion which one would expect from an aneurysm of this size at this place.

XII. The Congenital Anomalies of the Aorta

To understand the common anomalies of the aortic arch a few words concerning the embryology of this structure are necessary

The primitive aorta bifurcates into two ascending aortic roots from which six pairs of branchial arch arteries depart dorsally (fig 302) The branchial cleft arteries of each side join in two dorsal descending aortic roots which finally merge in a single descending aorta The branchial arch arteries are not all simultaneously present at any stage of embryonic life, some of them become transformed in different ways while others vanish more or less completely. The fourth branchial cleft arteries have special significance, the left is destined to form the ultimate aortic arch while the right antimere obliterates. Consequently one can regard the fourth branchial arch arteries as the primitive aortic arches Through the right and left, anterior and posterior aortic roots they establish the connection between the primitive ascending and descending aorta and form with them a vascular ring (fig 302) which may be designated the aortic ring.

This vascular ring, which embraces the trachea and esophagus, does not, to be

dysphagia lusoria (p. 418) only by the following fact: then, not only the right subclavian artery but the entire aortic arch prior to the departure of the subclavian artery changes over from the left into the right half of the thorax.

Bedford and Parkinson and Taussig and Grob suggest that arcus circumflexus is a displacement of the aortic arch to the right produced by a right-sided ligamentum arteriosum, a subclavian artery arising behind the esophagus as the last branch of the aortic arch, or, finally, a rudimentary persistent right aortic arch (arcus aortae duplex) (p. 413).

It is conceivable that a case of "low right position of the aorta" (Roesler and White, Pape) in which the descending aorta moves from left to right first at the level of or just below the bifurcation, owing to its nature, could not be distinguished from arcus aortae circumflexus sinister (fig. 273a and b). On this point, however, there are no reports to indicate whether these patients have a right-sided ligamentum arteriosum which might exert traction on the descending aorta. In this anomaly the distal part of the descending aorta, bending to the right, can project beyond the right border of the heart. At the site where the descending aorta crosses, the esophagus may show a circumscribed forward bend which is distinct in the right anterior oblique position.

2. *Arcus Aortae Duplex*

When the primitive aortic ring is not interrupted, both of the fourth branchial arch arteries form a right and left aortic arch from which arcus aortae duplex results (fig. 305a) In this anomaly the two aortic arches embrace the trachea and esophagus (fig 305b) like a clamp (in rare cases only the trachea is affected while the esophagus is located between the vessel ring and the spine), they may constrict these passages so that disturbances of breathing and swallowing result On the other hand, arcus aortae duplex may also remain completely asymptomatic when compression of the trachea and esophagus is very slight Each of the two aortic arches give off a common carotid and subclavian artery.

In the roentgenogram of this rare anomaly (Grob and Holzmann), corresponding to the relative narrowness of both arches, bilaterally and at the same height there is only a very flat aortic knob. In the esophagus there is an indentation from both sides (fig 305c). Since the two arches usually proceed obliquely rather than symmetrically in a ventrodorsal direction, the indentations of the esophagus are often more distinct on the left anterior and the right posterior borders if the ascending aorta is located on the right and the descending aorta on the left, however, the indentations on the esophagus are more distinct on the right anterior and the left posterior borders if the ascending aorta is located on the right and the descending on the left.

It is essentially the position of the descending aorta which determines the oblique course of the clamp-like aortic ring This course of the descending aorta is usually most obvious in the oblique positions since, when left sided, it can be demarcated in the left anterior oblique position and when right sided, in the right anterior oblique position within the vertebral shadow (p. 420). Likewise, the course of the barium filled retrocardiac section of the esophagus permits a conclusion about the position of the descending aorta, when left-sided the esophagus tends to bend

esophagus often shows a circumscribed bend to the left which is created by the application of the initial part of the descending aorta proceeding downward on the right. Subsequently, the esophagus moves to the left of the midline as it proceeds caudad. In the left anterior oblique position, the esophagus makes a marked turn

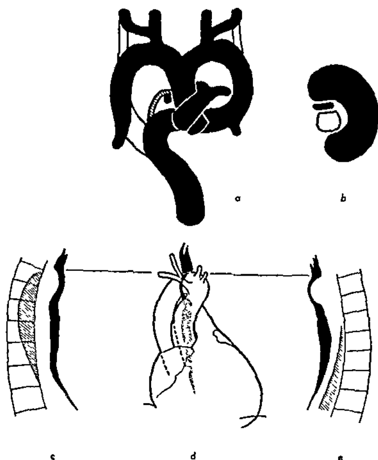


FIG 304.—(*a*) Genesis of arcus aortae circumflexus sinister with left sided ligamentum arteriosum. (*b*) Positional relation of arcus aortae circumflexus sinister to the trachea and esophagus (*c*) Course of esophagus and projection of the descending aorta proceeding caudad on the right in the spinal shadow in the right anterior oblique position (*d*) Posteroanterior projection with contrast filling of esophagus (trachea and superior vena cava omitted) (*e*) Course of the esophagus and projection of the descending aorta proceeding caudad in the retrocardiac field in the left anterior oblique position

forward as the result of the retroesophageal aortic arch while in the right anterior oblique position a somewhat lower, flat turn is created by the initial part of the descending aorta proceeding obliquely to the right caudad (Grob).

The departure of the brachiocephalic branches of the aortic arch may be normal with arcus aortae circumflexus sinister. There is an interesting variant in which the right subclavian artery departs as the last branch of the aortic arch after crossing the esophagus (Edwards). This variant is distinguished from the more common cause of

powerful aortic arch embraces the trachea and esophagus like an arcus aortae circumflexus from behind (figs. 307b and 308c).

Owing to these different courses, the slit formed by the vascular ring may take different directions as is indicated in figures 306b, 307b, and 308b and c Accordingly, the constriction which the esophagus and trachea experience from the ring vary and depend first, on which of the two arches is stronger and which rudimentary, and second, on whether the more powerfully developed aortic arch crosses the midline as an arcus circumflexus

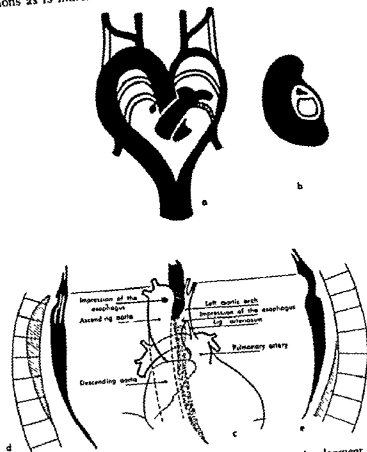


FIG 306—(a) Genesis of arcus aortae duplex with rudimentary development of left aortic arch (b) Positional relation of both aortic arches to trachea and esophagus (trachea and superior vena cava omitted) (c) Postero-anterior projection with contrast filling of esophagus (trachea and superior vena cava omitted) Right-sided indentation of esophagus by aortic arch proceeding on the right (↕), left-sided oblique indentation (↑) by the rudimentary left aortic arch (d) Dorsal indentation of esophagus in right anterior oblique position by the rudimentary aortic arch The descending aorta proceeding caudad on the right is discernible in the shadow of the spine (e) Ventral indentation of esophagus by the larger aortic arch proceeding on the right and a dorsal oblique impression by the rudimentary left aortic arch in the left anterior oblique position

In either instance, the ligamentum arteriosum lies more commonly on the left than on the right. It can definitely contribute to the constriction of the trachea and

somewhat to the right and when right sided, somewhat to the left (Zdansky, Thurnher)

Usually the two aortic arches are not equally developed. Rather, one is more or less narrow (Lockhart, Sprong and Cutler, Sweet and coworkers) (figs. 306a and 308a) or, in an extreme case, obliterated to form a solid cord (Arkin, Biedermann).

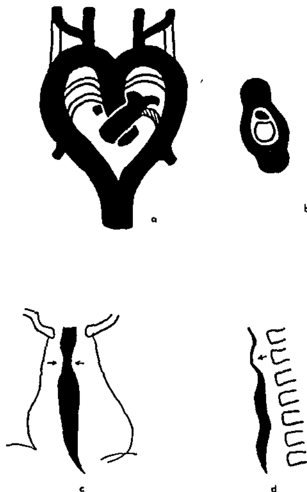


Fig 305 —(*a*) Genesis of arcus aortae duplex with both arches developed equally (*b*) Positional relation of two almost equally developed aortic arches to the trachea and esophagus. (*c*) Impression of the two aortic arches on the esophagus (*d*) Forward displacement of the esophagus at the level of the aortic ring

The more strongly developed arch can proceed over the left (fig. 308a) or the right (fig 306a) bronchus and often lies higher than the rudimentary arch. Right position of the stronger arch is more common than of the left. Moreover, their spatial relation ... high the descending ...ngly devel- ...us from the ...le, the more

(Gross, Sweet, Poth, Exalto et al). The roentgenologic determination as to whether a rudimentary aortic arch is present and the side on which it is located has, for this reason, special practical significance.

As a rule the position of the more powerfully developed aortic arch is distinct in the roentgenogram as the aortic knob, often this is located abnormally high and may extend to the level of the clavicle. The question about the side on which the descending aorta proceeds caudad can usually be decided in the oblique positions (figs. 306d and e and 307d and e). The rudimentary aortic arch is, on the whole, not

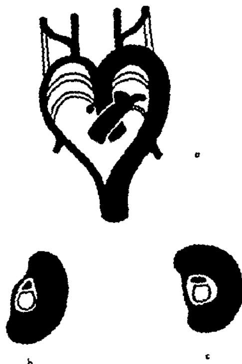


Fig. 308 — (a) Genesis of arcus aortae duplex with rudimentary right aortic arch ... tional relation of aortic arches to trachea and to esoph... (c) Positional relation of the aortic arches to trac... by arcus circumflexus sinister.

directly demonstrable or, at most it appears as a flat bulge on the side opposite to and usually somewhat lower than the aortic knob. In some cases, filling the esophagus with barium gives reliable information about the presence and the course of two aortic arches (fig. 306) On the side of the more strongly developed aortic arch there is a typical indentation (aortic bed) or even a circumscribed bend of the esophagus to the opposite side This circumscribed displacement may also be forward when the arch embraces the esophagus like an arcus circumflexus, this is most distinct in the oblique positions (fig. 307). The rudimentary arch can also produce an indentation in the barium-filled esophagus. While this is not always more shallow, owing to its lower position it is located more or less caudad to the larger aortic bed and, corresponding to the smaller diameter of the vessels, is smaller and is directed obliquely

esophagus when the rudimentary aortic arch, proceeding to the left, is drawn against the pulmonary artery by the inserted ligament (figs. 306c and 307c).

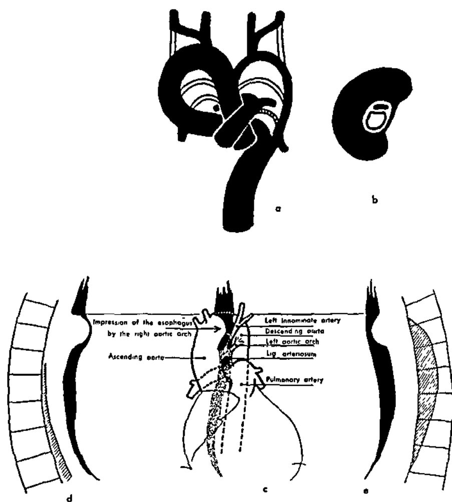


FIG 307 —(*a*) Genesis of arcus aorta duplex with arcus circumflexus and rudimentary left aortic arch (*b*) Positional relation of both aortic arches to trachea and esophagus. (*c*) Postero-anterior projection with contrast filling of esophagus (trachea and superior vena cava omitted). Indentation of esophagus from right and behind by the aortic arch proceeding on the right and drawing to the left behind the esophagus The rudimentary left aortic arch is drawn caudad and made tense by the ligamentum arteriosum (*d*) The descending aorta proceeding caudad on the left is visible in the right anterior oblique position in front of the spine in the retrocardiac space as a pale shadow. (*e*) In the left anterior oblique position it is visible as a darker shadow within the shadow of the spine

Division of the ligament by operation can immediately diminish such constriction (Exalto and coworkers).

The different variants of arcus aortae duplex may lead to life-endangering and fatal disturbances of breathing and swallowing, particularly in infants (Snelling and Erb, Wolman, Gross, Sweet and coworkers, Holzmann, Grob) and necessitate operative division of the constricting ring in the region of the rudimentary arch

(Gross, Sweet, Poth, Exalto et al). The roentgenologic determination as to whether a rudimentary aortic arch is present and the side on which it is located has, for this reason, special practical significance.

As a rule the position of the more powerfully developed aortic arch is distinct in the roentgenogram as the aortic knob, often this is located abnormally high and may extend to the level of the clavicle. The question about the side on which the descending aorta proceeds caudad can usually be decided in the oblique positions (figs. 306d and e and 307d and e). The rudimentary aortic arch is, on the whole, not

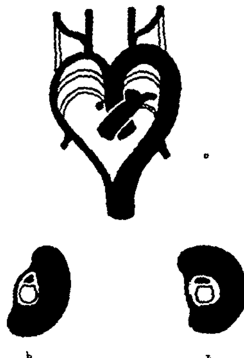


Fig 308 —(a) Genesis of arcus aortae duplex with rudimentary right aortic arch (b) Positional relation of aortic arches to trachea and to esophagus with the aorta descending on the left (c) Positional relation of the aortic arches to trachea and esophagus with aorta descending on right by arcus circumflexus sinister

directly demonstrable or, at most it appears as a flat bulge on the side opposite to and usually somewhat lower than the aortic knob· In some cases, filling the esophagus with barium gives reliable information about the presence and the course of two aortic arches (fig 306). On the side of the more strongly developed aortic arch there is a typical indentation (aortic bed) or even a circumscribed bend of the esophagus to the opposite side. This circumscribed displacement may also be forward when the arch embraces the esophagus like an arcus circumflexus, this is most distinct in the oblique positions (fig. 307). The rudimentary arch can also produce an indentation in the barium-filled esophagus. While this is not always more shallow, owing to its lower position it is located more or less caudad to the larger aortic bed and, corresponding to the smaller diameter of the vessels, is smaller and is directed obliquely

owing to the ascending course of the vessel (fig. 306). The constrictions of the trachea do not differ in principle from those of the esophagus but naturally they are not as deep nor as easily perceived.

A definite decision on whether the rudimentary aortic arch is actually pervious or obliterated to a solid cord sometimes can be determined by kymographic registration of the pulsations after filling the esophagus with barium, for this purpose aortography is useful if it is technically possible to enter the ascending aorta.

3 *Left Origin of the Right Subclavian Artery* (*Dysphagia Lusoria*)

An entire series of aortic malformations can occur when the interruption of the primary aortic ring does not develop at the proper place. One of the most common



FIG 309 —(*a*) Genesis of right subclavian artery arising on the left with retroesophageal diverticulum (*b*) Positional relation of the aortic arch, of the retroesophageal diverticulum, and of the right subclavian artery to the trachea and esophagus

anomalies of this kind emerges when regression of the right half of the primitive aortic ring occurs in the region of the fourth branchial cleft artery (fig. 309) rather than in the region of the descending aortic root. Under these circumstances the right common carotid artery represents a direct continuation of the right ascending aortic root while the right subclavian artery takes its origin from the descending aortic root. In the fully developed form the right descending aortic root often persists as a diverticulum-like rest from which the right subclavian artery arises as the last branch of the aortic arch proceeding on the left. Consequently the artery must cross from left to right behind the esophagus Occasionally the right common carotid artery



a



b



c

FIG 310—Right subclavian artery arising on the left (dysphagia lusoria) Female, about 50 years old At the upper border of the aortic arch the esophagus bends to the right and forward in a semicircle and here is somewhat narrow The radius of the bend showed rhythmic, systolic enlargement (*a*) Anterior view (*b*) Left anterior oblique view (*c*) Pathologic specimen of the case seen from behind

as well as the right subclavian artery departs from the aortic diverticulum. In such cases one must assume that the obliteration of the right half of the aortic ring occurred in the initial part of the ascending aortic root.

The anomaly was described by Bayford in 1794 as dysphagia lusoria and, according to anatomic statistics, occurs in 0.4 to 2.0 per cent of all necropsies. The artery proceeding to the right behind the esophagus or the diverticulum-like rest of the right descending aortic root from which the artery arises, *produces a characteristic* circumscribed turn, directed to the right and forward, and compression of the esophagus. In rare cases this may lead to dysphagia.

In 20 per cent of the cases the artery is not located behind the esophagus but rather between it and the trachea or even in front of the trachea (Holzapfel, Banchi); at present these variants are not satisfactorily explained from a developmental standpoint.

The roentgen findings are remarkably characteristic. Zdansky described the first case controlled by necropsy (fig 310a to c). Kommerell devoted an extensive publication to this common vascular anomaly. On films and with simple fluoroscopy, without filling the esophagus with barium, the roentgen picture shows no deviation from normal except at times a greater prominence of the aortic knob After filling the esophagus with barium it is noted that the esophagus describes a circumscribed, semicircular turn directed to the right and anteriorly on the upper circumference of the aortic knob (fig. 310a and b) This bend of variable size usually displays rhythmic systolic enlargement from which it can be inferred that it is produced by an arterial pulsating vessel inserted between the esophagus and spine Below this turn the esophagus often fails to show a typical aortic bed, rather it proceeds perpendicularly and straight to the left and below and, for this reason, Kreuzfuchs' measurement of the aorta is often impossible.

The radius of this bend in the esophagus is often very significant and permits the inference that a large diverticulum-like rest of the right descending aortic root forms the origin of the right subclavian artery.

From the standpoint of differential diagnosis one must always consider mediastinal glands or a retrosternal goiter. Rhythmic systolic enlargement of the semicircular bend dispels all doubt.

4 *Arcus Aortae Dexter (High Right Aorta)*

Not rarely, obliteration of the aortic ring occurs in the region of the left half The interruption may appear at different places Under these circumstances the right fourth branchial arch artery assumes the role of the persistent aortic arch. When the interruption takes place in the region of the left descending aortic root, distal to the subclavian artery (fig. 311), the brachiocephalic branches of the aorta depart in the following order· left innominate artery, right common carotid, right subclavian The descending aorta lies on the right side. If the interruption (and this is most common) occurs in the region of the branchial cleft arteries, proximal to the subclavian artery (fig. 312), a rudiment persists from the left descending aortic root and lies behind the esophagus to form a kind of diverticulum of the aortic arch which courses on the right. From this diverticulum the left subclavian artery departs.

Then, the brachiocephalic branches of the aorta have the following order left common carotid artery, right common carotid, right subclavian, left subclavian In rare instances, the left common carotid artery as well as the left subclavian arise from the retroesophageal diverticulum (Thurnher). This anomaly occurs when the left half of the aortic ring is interrupted directly after the division of the primitive ascending aorta. Exceptionally, the left common carotid artery arises from the diverticulum while the left subclavian artery is the first branch of the aortic arch (Holzmann).





Fig 311





Fig 312

Fig 311 —(*a*) Genesis of arcus aortae dexter (high right aorta) (*b*) Positional relation of aortic arch proceeding on right of trachea and esophagus.

Fig 312 —(*a*) Genesis of left subclavian artery arising on the right with arcus aortae dexter with retroesophageal diverticulum (*b*) Positional relation of aortic arch proceeding on the right and the left subclavian artery arising from the retroesophageal diverticulum to the trachea and esophagus

The diverticulum-like rest of the left part of the aortic ring is united to the pulmonary artery through the ligamentum arteriosum In this way a ring-like constriction of the esophagus and trachea may occur (Rossi and Rohner) although this is never as extreme as in *arcus aortae duplex* If the *ductus arteriosus is persistent*, in this way it may communicate with the left subclavian artery ("natural Blalock")

Arcus aortae dexter with the variants described occurs *relatively often* with Fallot's anomaly, in truncus and pseudotruncus arteriosus as well as in situs inversus

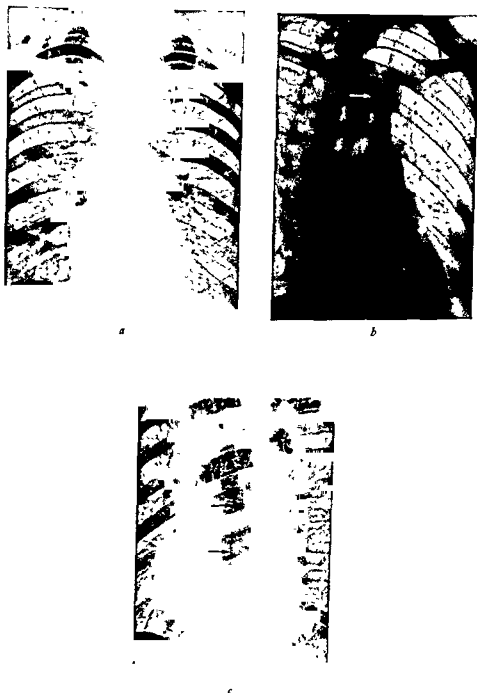


FIG 313 —Arcus aortae dexter. (*a*) Anterior view The aortic knob is missing The descending aorta forms the border of the right (*b*) Right anterior oblique view. At the level of the aortic arch is seen the diverticulum-like rudiment of the left posterior aortic root as a circular shadow (cut across by the arrow) and the trachea is indented from behind by this vascular vestige (*c*) Left anterior oblique view. One misses the shadow, ordinarily visible, of the distal division of the arch On the other hand, the shadow of the descending aorta on the right of the spine is easily seen far in front of the vertebral shadow (arrows).

abdominalis Biedermann found arcus aortae dexter seven times in five thousand examinations; Zdansky obtained the same result

The roentgenogram of arcus aortae dexter was originally described and confirmed at necropsy by Assmann. Since then innumerable papers have been published on this anomaly, of these the comprehensive works of Arkin, Kommerell, Grob, Franke, and Thurnher may be stressed Often the roentgen diagnosis can be made at the first glance when one misses the aortic knob on the left and sees a flat convex (fig. 313a, b and c) or actual knob-like projection (figs 314 and 315) below the right clavicle Filling the esophagus with barium ensures the diagnosis In the posteroanterior position the esophagus forms an aortic bed directed to the right and then proceeds caudad somewhat to the left of the midline (fig 315) Fluoroscopy



FIG 314 —Arcus aortae dexter Aortic lues, atheromatosis The aortic knob is seen on the right *The initial part of the descending aorta is noted on the right (white arrow), then it vanishes* behind the dilated and markedly calcified ascending aorta which bulges to the right (black arrow).

in the right anterior oblique position provides information about the presence or absence of a diverticulum-like rest of the left descending aortic root from which, as mentioned above, the left subclavian and sometimes the left common carotid artery as well, arise. If no diverticulum is present (fig 311) and the brachiocephalic vessels arise from the right sided aortic arch in an order the mirror image of normal, no displacement of the esophagus is seen in the right anterior oblique position (fig 316a and b) If, on the contrary, a vestigium of the left descending aortic root persists (fig 312), the esophagus bends forward at the height of the aortic arch (figs. 315c and 317b and c). This bend which often shows distinct systolic-expansile enlargement, increases in size in proportion to the size of the diverticulum and it is merely suggested when the subclavian artery simply proceeds to the left behind the esophagus Sometimes in this position one sees a slight indentation of the esophagus from in front, this happens *when a vestigium of the left aortic arch is present in the form of a pretracheal left subclavian artery or a solid cord Without aortography one* cannot always positively exclude *arcus aortae duplex with a rudimentary right* aortic arch

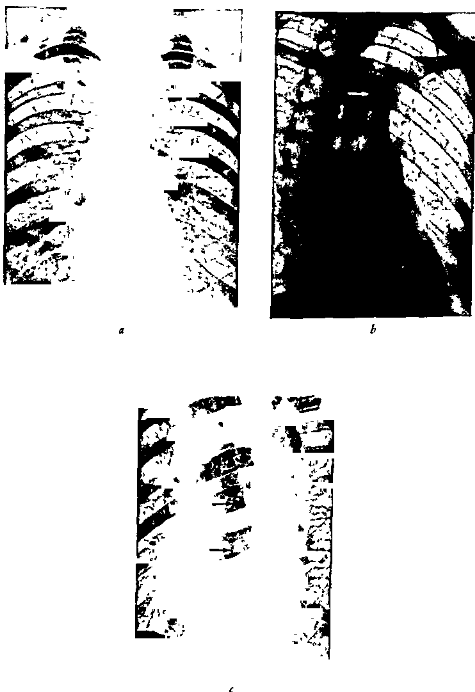


FIG 313 —Arcus aortae dexter (*a*) Anterior view. The aortic knob is missing The descending aorta forms the border of the right (*b*) Right anterior oblique view. At the level of

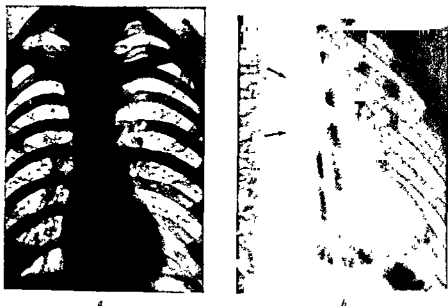


FIG 316 —Arcus aortae dexter without diverticulum-like rudiment of the left descending aortic root (a) Anterior view On the left side no aortic knob is present On the other hand, the descending aorta moves caudad on the right side (b) Right anterior oblique view The barium-filled esophagus shows only a very flat indentation of the posterior wall at the level of the aortic arch The descending aorta moving to the right from the midline is perceptible within the shadow of the spine (arrows)

5. *Arcus Aortae Circumflexus Dexter*

A very similar roentgenogram can occur when the aortic arch proceeding on the right—in analogy to arcus aortae circumflexus sinister (p 409)—bends to the left in an almost horizontal direction to proceed on the left side between the esophagus and spine and pass into an aorta descending on the left This arcus aortae circumflexus dexter (Kommerell) can give off the brachiocephalic branches in the same series as the arcus aortae dexter left innominate artery, right common carotid, right subclavian (fig 318). Accordingly, the left innominate artery must pass from the right-sided aortic arch, obliquely to the left and upward over the trachea The ligamentum or persistent ductus arteriosus lying on the left can join either the subclavian artery or the initial section of the descending aorta which lies on the left consequently to form a ring which embraces the trachea and esophagus on all sides. In other cases the left subclavian artery can pass on the left behind the esophagus as the last branch from the aortic arch This represents the mirror image of the same anomaly as arcus aortae circumflexus sinister (p 409) and corresponds to the retroesophageal left subclavian artery in arcus aortae dexter (fig 319) At times the sequence of brachiocephalic branches of arcus aortae circumflexus dexter may also be as follows right innominate artery, pretracheal left common carotid artery, and retroesophageal left subclavian artery (Taussig) From a roentgenologic standpoint all these variants have mainly theoretic interest since they cannot be distinguished from each other by x-ray



FIG. 315.—Arcus aortae dexter. (a) Anterior view with esophagus filled with barium The barium collects above the arch of the aorta and shows a circumscribed deviation to the left and forward. The right border of the descending aorta passes down from the flat aortic knob which is directed to the right. The esophagus proceeds along the left border of the spine and at the left border of the descending aorta (b) Right anterior oblique view without filling the esophagus with barium The trachea bends forward in an arc at the level of the aortic arch The descending aorta is well defined within the shadow of the spine. (c) Right anterior oblique view after filling esophagus with barium. The esophagus bends typically ventrad at the level of the aortic arch. This bend is produced by the diverticulum-like rest of the left descending aortic root.



a b

FIG 316—Arcus aortae dexter without diverticulum-like rudiment of the left descending aortic root (a) Anterior view On the left side no aortic knob is present On the other hand, the descending aorta moves caudad on the right side (b) Right anterior oblique view The barium-filled esophagus shows only a very flat indentation of the posterior wall at the level of the aortic arch The descending aorta moving to the right from the midline is perceptible within the shadow of the spine (arrows)

5. Arcus Aortae Circumflexus Dexter

A very similar roentgenogram can occur when the aortic arch proceeding on the right—in analogy to arcus aortae circumflexus sinister (p. 409)—bends to the left in an almost horizontal direction to proceed on the left side between the esophagus and spine and pass into an aorta descending on the left This arcus aortae circumflexus dexter (Kommerell) can give off the brachiocephalic branches in the same series as the arcus aortae dexter left innominate artery, right common carotid, right subclavian (fig. 318). Accordingly, the left innominate artery must pass from the right-sided aortic arch, obliquely to the left and upward over the trachea The ligamentum or persistent ductus arteriosus lying on the left can join either the subclavian artery or the initial section of the descending aorta which lies on the left consequently to form a ring which embraces the trachea and esophagus on all sides. In other cases the left subclavian artery can pass on the left behind the esophagus as the last branch from the aortic arch This represents the mirror image of the same anomaly as arcus aortae circumflexus sinister (p 409) and corresponds to the retroesophageal left subclavian artery in arcus aortae dexter (fig 319) At times the sequence of brachiocephalic branches of arcus aortae circumflexus dexter may also be as follows right innominate artery, pretracheal left common carotid artery, and retroesophageal left subclavian artery (Taussig). From a roentgenologic standpoint all these variants have mainly theoretic interest since they cannot be distinguished from each other by x-ray.

In all these cases by fluoroscopy one sees the esophagus bend forward and to the left at the height of the aortic arch just as this happens in arcus aortae dexter with a retroesophageal aortic diverticulum. Naturally this bend is of striking size since it is

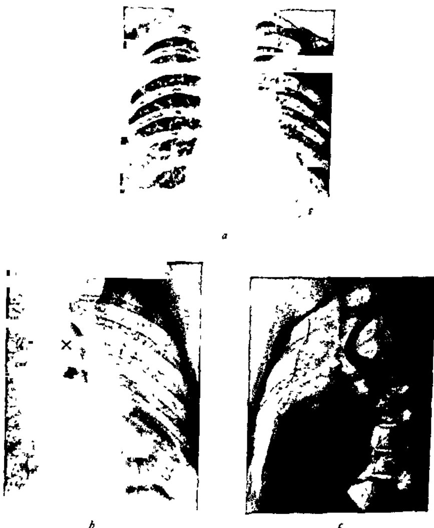


FIG 317 —Arcus aortae dexter. (*a*) Anterior view. The barium filled esophagus courses along the left border of the spine. (*b*) Right anterior oblique view. The esophagus and trachea bend forward at the level of the aortic arch. The x designates the shadow of the diverticulum-like rudiment of the left descending aortic root which produced this bend (*c*) Left anterior oblique view. In this position also a circumscribed bend of the esophagus is seen

produced *by the entire aortic arch which proceeds from right to left* behind the esophagus The demonstration of the course of the descending aorta is decisive in differential diagnosis In arcus circumflexus dexter its shadow is missed on the right side but is visible on the left below the clavicle and within the cardiac waist as a pale, slightly left-convexly curved and straight descending shadow. Fluoroscopy in the oblique positions is particularly informative. The descending aorta proceeding

on the left is seen in the left anterior oblique position ordinarily as a characteristic semilunar dark shadow within the spine and in the right anterior oblique position as a pale shadow in front of the spine within the retrocardiac space. The opposite situation prevails when the descending aorta proceeds on the right. Likewise, the course of the barium-filled esophagus provides good information on the position of the descending aorta. When the aorta descends on the left, the retrocardiac section of the esophagus deviates somewhat to the right while it turns somewhat to the left when the aorta descends on the right (Zdansky, Thurnher).









Fig 318 Fig 319

Fig 318 —(*a*) Genesis of arcus aortae circumflexus dexter (*b*) Positional relation of aortic arch to trachea and esophagus

Fig 319 —(*a*) Genesis of arcus aortae circumflexus dexter with retroesophageal diverticulum from which the left subclavian artery arises (*b*) Positional relation of aortic arch to trachea and to esophagus

It is evident that no decision is possible on the basis of the roentgenogram as to whether a rudimentary left aortic arch (p. 426) is or is not present in addition to arcus aortae circumflexus dexter This decision can be reached by aortography, although it is extremely difficult to enter the ascending aorta with the catheter

Complicated relationships exist in those cases in which the descending aorta, curving to the left, of an arcus circumflexus again turns into the right hemithorax, behind the esophagus, at or just below the tracheal bifurcation (Bedford and Parkinson, Roesler, Franke, Chiba, Seuderling and Kumlin) (fig 320). This S-shape in which the aorta crosses the spine twice is readily seen in hard films in the postero-anterior position and in films in the oblique positions. At first the esophagus is indented from behind and from the right at the level of the aortic arch and shows a displacement to the left and forward, then, at the level of or below the bifurcation it

experiences a new displacement forward since the descending aorta returns to the right. *Thereupon it follows the descending* aorta and is displaced ventrad once again in the supradiaphragmatic section as the aorta approaches the hiatus aorticus In all these cases as well as in one observed by us the aorta *showed considerable* dilatation Moreover it is probable that in some of these cases it is not a left sided

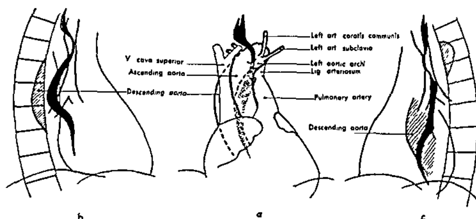


Fig 320 —(*a*) Arcus aortae circumflexus dexter with descending aorta proceeding caudad on *right and presumable rudimentary persistent left aortic arch (arcus aortae duplex?). Trachea* omitted Oesophagus filled with barium At the level of the aortic arch the esophagus is displaced to the left and forward Below this there is a smaller oblique impression to the left and forward presumably produced by a rudimentary left sided aortic arch Then, the esophagus proceeds to the right and downward with the descending aorta which finally displaces the esophagus once more to the front in the supradiaphragmatic area The last displacement is distinct in *b* and *c* (*b*) In the right anterior oblique position the descending aorta bending toward the right in the median section within the spinal shadow is visible as a thicker semilunar shadow. (*c*) In the left anterior oblique position one recognizes the descending aorta bending to the right within the cardiac shadow

ligamentum arteriosum which displaces the aortic arch to the left but a rudimentary aortic arch, then, in actuality an arcus aortae duplex would be present with arcus circumflexus sinister and an aorta descending on the right In a patient observed by Zdansky (fig. 320) these relationships were indicated by the course of the esophagus

6. *Coarctation of the Aorta (Isthmus Stenosis)*

By coarctation of the aorta one understands a narrowing, atresia, or complete interruption of the aorta in the region of the isthmus, that is, in the region of the insertion of the ductus or ligamentum arteriosum. In rare cases the actual stenosis is *located proximal to the left subclavian artery. Much more* often, the stenosis is located between the left subclavian artery and the insertion of the ductus or ligamentum arteriosum or at the site of the insertion In rare cases it may even be found in the section of aorta lying just distal to this area. Sometimes the aortic lumen is completely interrupted over a long section.

The "infantile type" of coarctation. In stenoses located *proximal to the* ductus arteriosus—between it and the left subclavian artery—the ductus usually persists so that the poststenotic part of the aorta is supplied from the pulmonary artery through

the patent ductus arteriosus. The designation as "infantile" type does not mean that it is limited to children for adults may be affected (Abbott) This form of coarctation is compatible with life only when sufficient arterialized blood passes through the stenosis or when the right ventricle pumps mixed blood rather than pure venous blood into the descending aorta, the latter situation is possible only with an intracardiac shunt such as an atrial or ventricular septal defect with or without a torsion anomaly of the bulbus-truncus section. As a matter of fact the infantile type is very often associated with congenital cardiac malformations by virtue of which life is possible for a time. No collaterals form No increase of blood pressure in the upper extremities develops. The electrocardiogram shows a right preponderance While the head and upper extremities obtain normal arterialized blood, the rest of the body receives mixed blood. For this reason the cyanosis has a striking inequality of distribution. Most of those bearing this anomaly die in early childhood

The roentgenogram is dominated by the cardiac anomaly usually present Corresponding to the augmented work of the right heart the cardiac shadow is usually somewhat enlarged and has a mitral configuration resulting from dilatation and hypertrophy of the right ventricle The pulmonary arc often protrudes decidedly into the cardiac waist. The stenosis can scarcely be directly visualized by fluoroscopy or films.

The "adult type" of coarctation When the stenosis is located at the isthmus, the ductus arteriosus is usually closed Adequate blood supply of the thoracic walls and the poststenotic part of the aorta is possible only by the development of collaterals which make their way from the brachiocephalic vessels over the internal mammary and intercostal arteries as well as over the arteries of the neck and shoulder girdle (fig 321a and b). The intercostal arteries, often enormously dilated and tortuous, produce erosions on the lower borders of the ribs which are important in diagnosis The collaterals often enlarge as the patient grows older Their pulsations are often palpable on the shoulder girdle and on the thoracic wall They appear particularly distinct when the patient bends forward with the arms hanging down (Campbell and Suzman). Cyanosis is absent. The blood pressure in the upper extremities is more or less elevated and is low in the lower extremities Capacity for exertion is not affected for a long time. H. Roesler and Lewis saw cardiac complaints develop only with military and war service Some patients complain of headache, cold feet, and delayed healing of injuries on the feet. Males are affected four times as often as females (Abbott). The prognosis is increasingly more favorable as the appearance of symptoms is postponed If circulatory disturbances already appear during youth, only rarely is the age of 30 exceeded.

Roentgen examination, particularly in combination with aortography, makes valuable contributions to diagnosis The cardiac shadow may be normal in size and shape (fig 322) but frequently it is moderately expanded to the left and of aortic configuration. Great enlargement is present only when an aortic valve insufficiency, a combined valve lesion (fig 323), or coronary sclerosis is present Then, the cardiac shadow shows the signs of an eccentric hypertrophy of the left ventricle. With failure of the left heart, mitralization and pulmonary congestion appear and finally enlargement of the right heart as well

The right border of the vascular band usually bulges more strongly to the right



a



b

Fig. 321.—Demonstration of collateral circulation by means of aortography in a patient with the adult type of coarcation (*a*) Opacification of the prestenotic portion of the aortic arch as well as of the dilated left subclavian and internal mammary arteries. (*b*) Opacification of the post-stenotic portion of the thoracic aorta over the intercostal arteries dilated as collaterals



a



b

FIG 322 —Coarctation of the aorta (*a*) Anterior view The cardiac shadow is normal in size and shape No aortic knob is present in this case (*b*) Left anterior oblique view. The shadow of the ascending aorta rises vertically The aortic arch and ascending aorta cannot be outlined The lower border of several ribs show erosions (arrows) which are very characteristic and are rarely absent

which may be explained by dilatation of the ascending aorta which often shows increased pulsations By contrast, Kjellberg and Rudhe observed electrokymographically on the poststenotic part of the aorta an abnormally flat systolic rise of the curve corresponding to pulsus tardus Sometimes the ascending aorta may show aneurysmal expansion.

In striking contrast to the usual widening of the ascending aorta, the aortic knob is often very small and flat; it may also be absent (fig. 322a)

The expansion of the brachiocephalic branches of the aorta result in widening the part of the mediastinum which lies above the aortic arch. More specifically, the greatly dilated left subclavian artery may project as an abnormal arch between the poststenotic part of the aorta and the clavicle so that a double aortic knob may be simulated (Schatzki, Klemola). This double arc is especially marked if either the left subclavian artery or the poststenotic part of the descending aorta or both are markedly dilated, if the ligamentum arteriosum is short, or if the coarctation is very narrow (Bruwer and Pugh). Stauffer and Rigler drew attention to visible pulsation below the left clavicle during fluoroscopy.



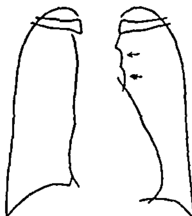


FIG 323 FIG 324

FIG. 323 —Coarctation of aorta with combined valvular lesions (necropsy). No aortic knob present Kreuzfuchs measurement cannot be made. On the lower borders of several ribs are typical erosions (arrows) At necropsy the stenosis which admitted a lead pencil was located just beyond the left subclavian artery.

FIG 324 —Coarctation with persistent ductus arteriosus. Female, 27 years old, with systolic-diastolic murmur over the pulmonic. Blood pressure in arms 165/80 mm. Hg and in lower extremities 115 mm Hg No abnormal pulsations on chest or back. No rib erosions Cardiac shadow not enlarged Over the protruding pulmonary arc, two smaller arcs, of these, the upper (↕) corresponded to the strongly pulsating prestenotic aortic arch and the lower (↕) to the weakly pulsating poststenotic dilated descending aorta (Case of Bramwell)

In the left anterior oblique position the shadow of the ascending aorta often climbs with striking sharpness to the summit of the vessel (Lewis). More important, however, is the distinct narrowing, step, or interruption of the aortic shadow often seen beyond the clear tracheal band The post stenotic part of the aortic arch often displays a knob-like widening. Occasionally calcium shadows are visible in the area of the stenosis. In younger subjects with a poorly defined aortic shadow the stenosis is often imperceptible.

An important sign, rarely absent in coarctation is the sharply outlined indentations of the lower borders of the dorsal sections of the ribs especially in the middle and lower parts of the chest (figs 322 and 323). The three uppermost pairs of ribs are almost never affected (Kirchhof and Lauer-Schmaltz). These alterations (H. Roesler, Railsback and Dock) correspond to Meckel's pressure-erosions from the dilated, tortuous intercostal arteries These erosions are rarely absent and are considered one of the most constant roentgen signs of coarctation (Ernstene and Robins,

K Weiss, Muller, Baker and Shelden). They are often absent in children but become more common, more numerous, and deeper with advancing years Laubry and Heim de Balsac correctly stress that the diagnosis of coarctation should not be made simply on isolated notches since the lower borders of the ribs are occasionally irregular for other reasons. In one case, Ratschow and Arendt observed rib erosions only on the right side, in this patient the coarctation was located above the departure of the left subclavian artery In a case of Franke, rib erosions were entirely absent, in this instance the aorta was narrow per se and the stenosis was located between the departure of the innominate artery and the left common carotid. The aortic knob was



Fig 325 —Hour-glass deformity in coarctation in a female, 21 years old Note the shortening of the aortic arch

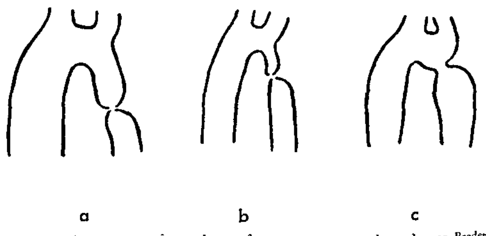
absent The trachea was displaced to the left by a greatly dilated and strongly pulsating innominate artery Death resulted from rupture of an aneurysm of a cerebral artery. There were also aneurysms at the orifices of the coronary arteries.

There are intermediate forms between the infantile and adult types of coarctation. Thus, the ductus arteriosus may be pervious despite a narrowing of the isthmus Then, the collaterals may be rather slightly developed but may still form if the ductus arteriosus is only narrow In one case of this kind (Bramwell) two flatly convex arcs were seen below the left clavicle, the upper showed marked, the lower weak pulsations (fig 324) A systolic-diastolic murmur over the pulmonic area as well as the conspicuous protrusion of the pulmonary arc indicated persistence of the ductus arteriosus The lower, weakly pulsating arc corresponded to the poststenotic dilated descending aorta while the upper, strongly pulsating arc was the dilated prestenotic aortic arch.

Apart from the differing sites and reciprocal relations to the brachiocephalic vessels, coarctation may show different widths, lengths, and course. These relations

interest surgeons since they have great significance in planning operation (Crafoord and Nylin, Gross and Hufnagel, Clagett, Blalock and Park). The most reliable information concerning these relations is provided by aortography in the left anterior oblique position. This should never be neglected prior to operation

Aortography reveals that, in general, the aortic arch tends to be shortened in coarctation (fig. 325). The aortic loop has lost its semicircular rounding and in the left anterior oblique position one observes slight downward kinking of the aortic arch after the departure of the innominate artery, more rarely, it is upward (Jonsson and coworkers) The hour-glass or transverse constricting stenosis may be located



at different distances from the departure of the left subclavian artery and the involved portion of the arch between this artery and the stenosis may vary in width and course. Jonsson and his coworkers have distinguished four types on a practical basis. 1) cases with a long and wide distal section of the aortic arch, 2) cases with a long and narrow distal section of the aortic arch which may sometimes bend ventral, 3) cases with a short distal section of the arch; and 4) cases with complete interruption of the aortic lumen (fig. 326). According to Crafoord, type 1 gives the best prospect for end-to-end anastomosis. In type 2, the results of operation are definitely less favorable and it is often necessary to employ a free vascular transplant (Gross) or to use the left subclavian artery to effect a junction (Clagett, Blalock and Park). In type 3 the proximity of the subclavian artery creates operative difficulties

Chapter Eight

The Pulmonary Artery and Its Pathologic Alterations

The pulmonary artery starts on the left, in front and somewhat above the aorta; it rises in a gentle curve to the left, more or less vertically and posteriorly to embrace the left wall of the ascending aorta by its close application Consequently, the right border of the pulmonary artery is not visible in any position Only the left and anterior boundaries can be demonstrated roentgenologically. The first forms the pulmonary arc in anterior views, the latter is visible for a short distance in the right anterior oblique and lateral position above the flat curve of the conus pulmonalis According to the position of the heart in the chest, the length and curvature of the pulmonary arc varies Descent of the diaphragm tends to elongate and stretch it while elevation shortens the pulmonary arc and produces greater bulging

The demonstrability of both branches of the pulmonary artery and their branches in anterior and oblique views was discussed on pages 25ff, 32, and 41ff

The width of the pulmonary trunk is not susceptible to measurement Assmann proposed to measure the width of the pulmonary artery by the transverse diameter of the right hilar shadow on films with 150 cm focal distance. He assumed that the shadow descending from the right lung root corresponded essentially to the main branch of the pulmonary artery after some branches departed for the upper lobe Measurement was made just below the downward curve of the hilar shadow. It fails when an enlarged heart overlaps the hilus or when the hilar shadow has faded outlines owing to pulmonary congestion. In adults the normal breadth of the hilus is 11 to 14 mm (average 13 mm) on films of 150 cm focus-film distance It varies with age, size, and weight. According to Assmann, values above 15 mm at least express pathologic dilatation in the lesser circuit (mitral lesions, emphysema, atrial septal defects, open ductus Botalli, pulmonary sclerosis, and so forth) Assmann found values of 9 to 10 mm. primarily in pulmonary stenosis. Naturally the breadth of the hilus does not always parallel the width of the trunk of the pulmonary artery. Thus, the trunk can be dilated while both branches are normal in caliber or narrow (in pure

pulmonary stenosis, for example) and, reversely, only one branch of the pulmonary artery need be dilated while the trunk and other branch are normal in width

In measurements of the hilus, enlarged hilar lymph nodes and infiltrates must be excluded since they may widen hilar shadows.

Demonstration of the larger ramifications of the pulmonary artery and of the pulmonary veins may be made by tomography. The distribution and caliber of the great branches is shown well when the surrounding lung tissue contains air and no inflammatory or neoplastic infiltration is present and atelectasis is absent.

In this way, atypical courses of large pulmonary veins can be shown clearly (fig. 327). Then, one sees on the right, more often than on the left, the pulmonary



FIG. 327 —Atypical veins in the right lung which unite with a large trunk in the base of the right lung and of which one branch proceeds to the left atrium and the other goes through the diaphragm into the abdomen The patient was clinically free from complaints (tomogram).

veins converging on abnormally proceeding trunks which advance in an arc to the lower, more rarely to the upper, venous infundibulum or can even be followed caudad to the diaphragm (Dotter, Hardisty, and Steinberg). Often these veins pass through the diaphragm and may empty into the inferior vena cava or hepatic veins For the most part, these anomalies are incidental findings and devoid of practical significance.

To demonstrate the finer vessel branches and the vessels in infiltrated or indurated lung tissue, opacification by contrast agents is indispensible (p 13). Angiography of the lungs was initially undertaken for diagnostic purposes by Forssmann as well as by Moniz, de Carvalho, and Lima Subsequently the technic was developed by Castellanos, Pereira, and Garcia, and particularly by Robb and Steinberg. Zorn introduced opacification of single segmental arteries and veins by opaque material through a cardiac catheter. A description of the findings in various

diseases of the lungs, in particular in tumors, tuberculosis, cysts, silicosis, bullous emphysema as well as in mediastinal tumors, is not germane to the present discussion

I. Dilatation of the Pulmonary Artery

In the anterior view, dilatation of the main stem of the pulmonary artery causes the pulmonary arc to project into and more or less fill the cardiac waist. Otherwise the cardiac shadow may be entirely normal. In some cases, however, its size and shape are definitely influenced by the cardiac and circulatory dynamic conditions responsible for dilatation of the artery, then, as a rule, right ventricular hypertrophy and dilatation is found

Ordinarily dilatation is not limited to the trunk of the pulmonary artery but extends to its branches. Consequently, in most cases the vascular shadows in the lungs (hilar shadows and arterial lung markings) are accentuated In pulmonary stenosis, to be sure, only the trunk of the pulmonary artery may be dilated while the intrapulmonic vascular structures are abnormally delicate and sparse In persistent ductus arteriosus there is often dilatation only of the trunk of the pulmonary artery and of the left branch with corresponding enlargement of the left hilar shadow.

On the prominent pulmonary arc and the enlarged hilar shadows, one often sees systolic expansile pulsations which may be very distinct when cardiac action is excited and the right ventricle is hypertrophied With a large pulse pressure the pulsations may resemble pulsus celer

In the right anterior oblique position the dilated pulmonary artery trunk bulges distinctly. The cross section of the left branch is easily recognized as a dark round shadow in the base of the vascular band (fig. 17a and b, item 15)

In this position the esophagus often bends to the right and backward below the aortic bed Parkinson and Bedford ascribed this to dilatation of the pulmonary artery. Since the bifurcation of the trachea and left main bronchus is interposed between the artery and esophagus, presumably this bend or indentation is not produced directly by the dilated artery but by the left main bronchus which is displaced by the dilated pulmonary artery to press against the esophagus

If conditions are otherwise favorable, in the left anterior oblique position the widened band of the left pulmonary artery crosses the retrocardiac field transversely below the aortic arch Branches of the right pulmonary artery are visible below the vascular band

Occasionally, in the sagittal (p-a) and oblique positions, calcium deposits are noted in the wall of the trunk (fig 328) or the larger branches (Rosler, Kreuzfuchs)

A distinction can be made between dilatation produced by the anlage and the acquired form. The first usually depends upon unequal division of the truncus arteriosus as reported by Laubry, Routier, and Heim de Balsac. Some bulges of the pulmonary arc in youthful individuals and in patients with thyrotoxicosis have been improperly interpreted in this sense. Oppenheimer and Taussig each described an instance of extreme idiopathic dilatation of the pulmonary artery and its large branches. Taussig's patient was a child in whom the dilated pulmonary artery compressed the trachea and bronchi leading to severe dyspnea and cyanosis. Zdansky

observed monstrous dilatation of the pulmonary artery and its intrapulmonic branches in a man with retinitis pigmentosa.

Acquired dilatation of the pulmonary artery is decidedly more common. Usually there is a dynamic dilatation, or one originally dynamic and later anatomically fixed, the causes of which *may be congenital or acquired*. *Almost invariably this* dilatation results from increased intra-arterial pressure, much less often it depends upon an acquired vessel injury and only exceptionally is an inborn distensibility and thinness of the vessel responsible (Esser).

Among the acquired dilatations are those in mitral lesions, acquired diseases of the pulmonary valve, emphysema, pulmonary sclerosis, pleural scars, and other conditions accompanied by increased resistance in the pulmonary circuit. If this



Fig 328 —Atheromatous calcium deposits in the trunk of the pulmonary artery (arrow). Right anterior oblique view

heightened resistance is only transient, dilatation of the pulmonary artery may recede as long as it is dynamic. Thus, protrusion of the pulmonary arc from failure of the left heart not rarely vanishes as cardiac function improves. Assmann also observed this in diphtheria and Forster in paroxysmal tachycardia. For other reasons, dilatation of the pulmonary artery capable of recession, is seen in beriberi (Reinhold, Aalsmeer and Wenckebach). Dilatation of considerable and even aneurysmal dimensions in some cases of mitral stenosis should not be considered *merely the effect of increased pressure but also of rheumatic arterial damage* (Kugel and Epstein, Chiari, Laubry and Thomas).

Dilatation of the pulmonary artery, like that of the aorta, does not parallel the pressure prevailing in it, thus, the artery need not dilate demonstrably despite severe emphysema.

When no clinical or roentgenologic signs of a congenital cardiovascular anomaly or any lesion elevating pressure is present, the possibility of a luetic vascular lesion, syphilitic pulmonary arteritis, must be considered (Neuburger, Posselt) to explain the widened artery in a young person, congenital weakness of the wall must also be suspected (Esser).

Dilatation of the pulmonary artery as the result of contracting lesions in the left upper lobe or from left pleuromediastinal scars is extremely common Traction may cause the pulmonary artery to project into the cardiac waist angularly or like a gibbus (fig. 159). The distortion may cause valvular incompetence so that relative pulmonary regurgitation occurs (Neumann, Scherf). Often dilatation of the artery is merely simulated by the retraction.

Aneurysmal dilatation of the pulmonary trunk may be diffuse and fusiform or show a more saccular form Important summaries are those of Boyd and McGavack as well as Deterling and Clagett Usually the fusiform type is associated with greater dilatation of the large main branches, the saccular form is more circumscribed and is less common Aneürysm of the trunk of the pulmonary artery is found with mitral lesions, with and without pulmonary regurgitation, in various forms of pulmonary sclerosis (Arrilaga, Dietrich, Ludin and Kappeli), in lues (Plenge, Posselt, A Vogl, Luisada), and in congenital cardiovascular anomalies or without ascertainable cause At all events, more than one third of the cases develop on the basis of syphilis (Deterling and Clagett). Occasionally the aneurysm leads to a stenosis of the left main bronchus (Ludin and Kappeli). In the roentgenogram one sees a spherical or oval shadow which tends to project to the left and forward from the cardiac waist It rests on the mediastinum on a broad base and is sharply in contrast to the lung providing the latter is not atelectatic nor obscured by blood seeping into the pulmonary parenchyma with beginning perforation. Occasionally, mural calcifications are visible (Deterling and Clagett). Usually systolic-expansile pulsations are present These may be absent, however, when the wall of the aneurysm is lined by parietal masses of coagulum. In this way, a solid structure may be simulated Consequently, confusion with tumors of the hilar glands and of the mediastinum is common (Wahl and Gard).

The differentiation of an aneurysm of the pulmonary artery from one of the ascending aorta developing to the left often is not possible with certainty Large pulsating hilar shadows as well as signs of dilatation and hypertrophy of the right ventricle speak in favor of a pulmonary artery aneurysm in dubious cases Strong pulsations of a dilated pulmonary artery result in rare cases from rupture of an aortic aneurysm into it (fig. 285)

Left sided solid and cystic mediastinal tumors and enlarged hilar lymph nodes may simulate pulmonary artery aneurysm Even unequivocal systolic expansile pulsations are not definite proof of an aneurysm since tumors of hemangiomatous structure or metastatic hypernephroma may also show pulsations (fig 284) and tumors which grow around the pulmonary artery can transmit pulsations which cannot be distinguished from intrinsic ones, Zdansky saw this repeatedly in lymphogranuloma A definite distinction of these processes from an aneurysm is often possible only by angiocardiography (Dotter and Steinberg)

Sometimes an aneurysm of the ascending aorta may simulate an aneurysm of the pulmonary artery by displacing the latter If the trunk and left pulmonary artery are compressed in this way, the left hilar shadow and vessel markings of the left lung may be distinctly reduced (fig 296)

Apart from small aneurysms in tuberculous cavities, aneurysms of the peripheral branches of the pulmonary artery are rare Usually they are mycotic or from

rheumatic endarteritis. Roentgenologic literature, moreover, contains descriptions of isolated traumatic aneurysms after gunshot wounds of the lungs (Marbleu, White). In all cases, round or oval, solitary or multiple, usually systolic expansile shadows are found at the hilus or along the vessels. They may develop in a week (Wedler) but they may also enlarge slowly (Weise). Zdansky saw multiple, cherry-sized mycotic aneurysms on branches of the right pulmonary artery as pulsating shadows arranged like a string of pearls in a patient with ulcerative endocarditis (fig. 329). Arteriovenous aneurysms of the lungs, usually congenital and rarely mycotic or



FIG 329 —Mycotic aneurysms of right pulmonary artery (necropsy). Male, 27 years old, with subacute bacterial endocarditis. On the lower pole of the right hilus are two round, cherry-sized, sharply defined shadows which showed systolic-expansile pulsation.

traumatic, provide a very characteristic picture which was described on page 135. Recognition is extremely important since they may endanger life by hemorrhage. Roentgenologic discovery alone affords the possibility of the only effective treatment, namely, removal of the affected lobe or segment.

In addition to the round shape, the systolic expansile pulsations of the intrapulmonic shadow are very characteristic of its aneurysmal nature. With thrombosis in the aneurysm, however, these pulsations can be absent (Wedler). Then, no absolute differentiation from hilar or parahilar glandular tumors, primary or metastatic lung tumors, round tuberculous foci, cysts, chondroma, or echinococcus is possible.

Varicose pulmonary phlebectasia must also be taken into consideration in differential diagnosis. Only five examples of these extremely rare and presumably congenital formations have been described. The first case (Hedinger) was not examined roentgenologically; the second (Nauwerck) had no roentgen findings, the remaining

three cases (Klinck and Hunt, Neiman, Jacchia) showed solitary round, sharply outlined shadows Only once was it thought that pulsations were visible. In one case the shadow gradually increased in size and finally resembled an orange In another (Jacchia) a small band ran from the round shadow to the heart, corresponding to a vein striving to reach the left atrium. In this case it was merely an incidental finding, in all other cases rupture of the varix was the immediate cause of death Despite its rarity, it concerns a finding of practical importance Furthermore, it is probable that most phlebectasias of the lungs are a sequel and partial manifestation of an arteriovenous fistula and consequently belong in the chapter on arteriovenous aneurysms (p. 135). At all events a distinction is not possible roentgenologically

II. Persistent Ductus Arteriosus Botalli

Ductus arteriosus Botalli is the fetal connection between the pulmonary artery and aorta Usually its opening is not located directly where the pulmonary trunk bifurcates but somewhat beyond the departure of the left branch (Hyrtl, Zuckerkandl, Langer) This is important for understanding some roentgenograms of persistent ductus Botalli

Normally the ductus closes in the first two weeks of life to form the Chorda Botalli which, when appropriately long and with otherwise favorable conditions, is visible in the left anterior oblique position (fig 25b) as a shadow bridging the pulmonary artery and aortic arch

Usually a persistent ductus is a cylindrical tube of varying width and length, it may be 2 cm long or longer Sometimes there is no tube but a mere opening through which the arteries in apposition communicate. In other instances the canal looks like a funnel whose wide end opens toward the aorta Furthermore, the canal may show an aneurysmal dilatation Sometimes the connection is not patent but is closed at its end in the pulmonary artery or, more rarely, in the aorta

In general, with uncomplicated ductus Botalli apertus, owing to higher pressure in the aorta, blood passes from it to the pulmonary artery which may dilate from abnormal increase of pressure From this systolic overloading follows either pure hypertrophy or an elongation and hypertrophy of the right ventricle which may lead to a mitral configuration In respect to the aorta, the abnormal discharge of blood causes a decided fall in diastolic pressure and a greater pressure amplitude which is expressed in pulsus celer, sometimes this is demonstrable in the radial artery Moreover the left ventricle becomes dilated and hypertrophic from diastolic overloading since it receives from the lungs not only the normal quantity of blood but also the blood shunted through the patent ductus arteriosus from the aorta into the pulmonary artery Naturally all these changes can be expected only when the open ductus is relatively wide

Persistent ductus arteriosus is not very common as an isolated anomaly, usually it is a single manifestation of major anomalous formations of the heart and sometimes makes possible the maintenance of life by creating a connection between the two circulations.

Uncomplicated persistent ductus arteriosus is two to three times as common in females as in males. Primarily, it has practical importance because those afflicted with this anomaly frequently develop subacute bacterial endocarditis and because further myocardial damage in these hearts is tolerated less well than in normal ones since an abnormal load has already been created by the short-circuit This justifies surgical intervention in certain cases whereby the abnormal vascular communication is closed (Gross and Hubbard)

Clinically, a continuous systolic-diastolic murmur accompanied by a corresponding palpable thrill is usually found in the second left anterior intercostal space, it is transmitted to the vessels of

the neck and to the back Sometimes a thrill of the aortic arch is palpable in the jugulum The second pulmonic sound is accentuated and often palpable. In children and young subjects no murmur may be audible or a pure systolic or completely noncharacteristic one may be heard With a very wide or very narrow ductus arteriosus, all murmurs may be absent in an adult. A very similar continuous systolic-diastolic murmur may be heard in aortic stenosis and regurgitation, in rupture of an aneurysm of the sinus of Valsalva into the right ventricle, or in a congenital defect of the truncal septum.

With persistent ductus arteriosus, pulsus celer may be present in the radial artery Cyanosis and clubbed fingers do not occur in the absence of other anomalies.

The roentgenogram of the heart may be normal in uncomplicated ductus arteriosus persistens, when the shunt is trifling. If, on the other hand, sufficient blood is driven into the pulmonary artery under aortic pressure, dynamic dilatation of the pulmonary artery may result with protrusion of the pulmonary arc into the cardiac waist. Often the heart is moderately enlarged to the left and shows elongation and greater rounding of the left ventricular arc. The posterior wall of the heart may show greater backward bulging and the esophagus may be displaced dorsad in a limited area (Pugh). These alterations are the expression of dilatation and hypertrophy of the left heart as the result of greater filling arising from the additional blood introduced from the pulmonary circulation through the persistent ductus. Furthermore this dilatation may clearly recede within a few weeks after the surgical elimination of the short-circuit (Taussig)

Pulsations like those of pulsus celer are often seen on the aortic knob as the result of the marked fall of diastolic pressure in the aorta Augmented pulsations on the pulmonary arc and of the hilar shadows are observed as the result of the added systolic influx of blood.

Often, however, all these signs are not very striking. Marked bulging of the pulmonary arc, decided dilatation, and pulsation of the intrapulmonic vascular shadows may even awaken the suspicion of the presence of some other anomaly such as an atrial septal defect.

Occasionally only the left hilar shadow is enlarged and markedly pulsating (Hochstadt and Silbermann, Zdansky). This isolated dilatation of the left pulmonary artery speaks in favor of the influx of blood mainly into the left pulmonary artery. Actually Dotter and Steinberg observed in angiocardiograms that the left pulmonary artery may be filled more than the right through a persistent ductus arteriosus

In the left anterior oblique position occasionally the persistent ductus arteriosus is directly visible as a band between the pulmonary artery and the aorta. Within the ductus or at its orifice in the aorta at times calcifications are seen and may serve in the roentgenogram (fig 330) as an indication that this anomaly is present (Abbott, R. White, F Weiss).

Generally speaking, the roentgenogram of a persistent ductus arteriosus is not very characteristic Similar and identical pictures occur in thyrotoxicosis, pulmonary regurgitation, septal defects, cor pulmonale, and in perforation of an aneurysm of the sinus of Valsalva into the right ventricle, and so forth

Thereby, angiocardiography obtains special significance in the roentgenologic study of clinically doubtful cases With sagittal projection one may sometimes perceive a small filling defect immediately after opacification of the right heart and the pulmonary artery at the dome of the pulmonary artery shadow. This develops owing to the entrance of aortic blood, not containing opaque material, through the ductus

(Goetz). Furthermore, one can demonstrate abnormally long opacification of the lesser circuit and of the left heart which outlasts opacification of the right heart for some time. This is explained by the refilling through the persistent ductus

In the left anterior oblique position one can see directly the passage of opacified blood from the aorta into the pulmonary artery This is achieved best by means of aortography. In this way one may determine the position, approximate width, and length of the ductus in some cases and this is important in regard to surgical intervention (Jonsson, Brodén and Karnell) Great caution is in order in roentgenologic



Fig 330 —Mural calcifications of aorta at the opening of a persistent ductus Botalli (arrow) (necropsy)

1 Arch of aorta
2 Calcifications in aorta at opening of a persistent ductus arteriosus (arrow)
3. Ductus arteriosus
4 Left pulmonary artery
5 Descending aorta

evaluation of the length and diameter of a persistent ductus On one side one must consider the exaggeration through central projection and, on the other side, the fact that the diameter of the ductus seems greater and its length shorter than corresponds to its open space. Jonsson and coworkers obtained a satisfactory agreement with the anatomic situation when they subtracted 2 mm from the roentgenologic diameter of the ductus and added 2 to 3 mm to the roentgenologic length Aortography confirms that less blood appears in the pulmonary artery through a narrow ductus than through a wide one but it also shows that a wide ductus is not always associated with a great shunt In recent years an increasing number of reports have appeared in which a wide persistent ductus was not associated with a shunt or with murmurs

Frequently at the orifice of the ductus arteriosus one sees a circumscribed dilatation or funnel-like, pointed, or shell-like bulge on the aorta (Steinberg, Grishman and Sussman, Jonsson and coworkers). This diverticulum may be located at the

anterior or antero-inferior border of the aortic arch. It is visualized best in the left anterior oblique and in the left lateral position. Moreover one may see a larger diverticulum below the aortic knob with sagittal projection as well. These formations appear, however, also in the absence of a persistent ductus arteriosus (Dotter and Steinberg, Wegelius).

On the basis of these diverticulum-like formations, an aneurysm can appear at the orifice of the ductus in the aorta and it may owe its development to a gradual dilatation of this weak point in the aortic tube. In favor of this is their appearance in the first few months of life (Scheef, Traum). On the other hand one must consider their mycotic origin (Eppinger) since sepsis and inflammatory lesions are very common in this anomaly All these aneurysms may lead to a paresis of the recurrent laryngeal nerve. They may rupture into the lungs, pleural or pericardial cavities.

Aneurysms of the partly obliterated ductus are more common. In this case there is an aneurysmal dilatation of the part of the ductus connected with the aorta while the orifice in the pulmonary artery is obliterated. Graham described an aneurysm of this kind, almost the size of a fist, with a visible calcium shell which protruded far into the left lung field with displacement of the aorta and trachea.

Chapter Nine

Lobus Venae Azygos (Wrisbergi)

Lobus venae azygos is the result of an abnormal vascular development in which the azygos vein arising from the supracardiac vein does not empty directly into the right ductus Cuvieri but, by a detour, into a partially

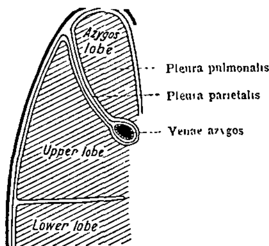


Fig. 331.—Schematic frontal section through lobus venae azygos

persistent cardinal vein (Lutz) An accessory lobus venae azygos might also develop by virtue of the part of the azygos arising from the persistent right cardinal vein not contacting the superior vena cava within the mediastinum above the right main bronchus but embedded in a deep notch in the right upper lobe, then it would pass to the superior vena cava through the posterior mediastinum (Lutz). The part of the lung separated from the upper lobe in this way is connected with the root of the lung by a fairly thin stalk which may differ greatly in size The notch also varies greatly in depth It is curved laterally convex in a ventrodorsal and craniocaudal direction. The vena azygos lies in its depth and has a cephalic and laterally convex course in a sort of mesentery formed by the parietal pleura (fig 331)

anterior or antero-inferior border of the aortic arch. It is visualized best in the left anterior oblique and in the left lateral position. Moreover one may see a larger diverticulum below the aortic knob with sagittal projection as well. These formations appear, however, also in the absence of a persistent ductus arteriosus (Dotter and Steinberg, Wegelius).

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The roentgenogram is readily understood from the course of the accessory interlobar cleft, this was originally described and correctly interpreted by Velde and was subsequently confirmed at necropsy by Bendick and Wessler as well as by Leeser.

In the anterior view (fig. 332) a hair-like line, curved laterally convex, usually divides the apex and adjoining infraclavicular field into two parts. This line corresponds to a notch which separates the accessory lobe from the upper lobe Its anatomic substrate is the mesentery-like reduplication of the parietal pleura which descends into a deep cleft and two layers of visceral pleura which cover the upper lobe and the accessory lobe, these face each other.

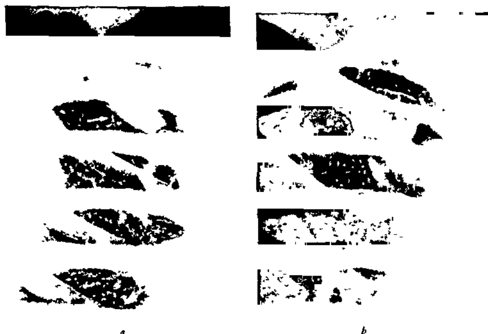


FIG. 334—Lobus venae azygos (*a*) In the anterior view the azygos vein, proceeding interlobar, projects from the mediastinal shadow like a twisted band (arrow) (*b*) With slight rotation to the right the spindle-shaped diagonal projection of the vein is seen

The lung median to the delicate line corresponds to the lobus venae azygos, frequently it is less clear than the rest of the upper lobe

According to the size of the accessory lobe, the upper end of the cleft lies either on the median, upper, or lateral boundary of the apex, more rarely it lies below the clavicle (fig. 333a). Often the shadow takes its origin from a pointed extension of an accompanying shadow to the second rib (fig. 334b) Below, it passes into a globular thickening near the upper pole of the hilus and advances directly into the mediastinal shadow This drop-like thickening is produced by the vein encircled by connective tissue covering it as far as it runs, approximately in the path of the central ray or somewhat obliquely. Often from the drop-like shadow, the band of vein can be followed median and caudad to the mediastinal shadow (fig. 333a) (Hjelm and



FIG. 332 —Lobus venae azygos. One sees how the hair-like line passes below the clavicle into a fusiform shadow of the vein (arrow) encountered in the path of the central ray. From this place the vein passes median and downward toward the mediastinal shadow.



FIG. 333 —Lobus venae azygos (*a*) Anterior view (*b*) With slight rotation to the right, the shadow of the vein (median arrow) emerges from the mediastinum

Bibliography

A

Aalsmeer, W. C., and Wenckebach, K. F. Herz und Kreislauf bei der Beri-beri-Krankheit Wien Arch f inn Med, *16* 193, 1929

Abbott, M. E. Coarctation of the aorta of the adult type Am Heart J, *3* 574, 1928.

Abbott, M E. Atlas of Congenital Cardiac Disease New York, Am Heart Assoc, 1936

Abbott, M E., Lewis, D. S, and Beattie, W W Differential study of a case of pulmonary stenosis of inflammatory origin (ventricular septum closed) and two cases of (a) pulmonary stenosis and (b) pulmonary atresia with associated ventricular septal defect and death from paradoxical cerebral embolism. Am. J M Sc, *165* 638, 1923

Abrams, H L, Kaplan, H S, and Purdy, A Diagnosis of complete transposition of the great vessels Radiology, *57* 500, 1951

de Abreu, M Études Radiologiques sur le *Poumons et le Mediastin* Paris, Masson, 1930

de Abreu, M Rontgengeometrie des Mediastinums Fortschr a d Geb d Rontgenstrahlen, *51* 564, 1935

de Abreu, M. Differentialdiagnose von Geschwulsten der *Aorta thoracalis* (auf Grund der Hydrodynamik) Fortschr. a d. Geb d Rontgenstrahlen, *51* 574, 1935

Achelis, W. Zur orthodiagraphischen Darstellung der Herzspitze Munchen med. Wchnschr, *57* 2225, 1910

Achelis, W Ueber adhasive Perikarditis und uber den Verlust der beim Uebergang aus der horizontalen zur aufrechten Korperstellung normalerweise eintretenden Vertikalverschiebung des Herzens, Dtsch Arch f klin. Med, *115* 419, 1914

Ackermann, R Beobachtungen uber die Veranderungen der Herzgrosse, der Puls- und Atemfrequenz und des Blutdruckes nach maximaler Laufleistung. Ztschr f klin Med, *103* 800, 1926.

de Agostino, P Orthodiagraphische und radiographische Untersuchungen uber die Grossenanderungen des Herzens in Beziehung zu Anstrengungen Ztschr exper Path u Ther, *7* 159, 1910

Ahlberg, N Ein Fall von traumatischer Herzklappenruptur Acta paediatr, *28* 69, 1940

Åkerlund, A Zur Frage der "reponiblen Hiatushernien." Dtsch med Wchnschr, *58* 1713, 1932.

Åkerlund, A, Ohnell and Key, E Hernia diaphragmatica hiatus oesophagei Acta radiol (Schwd), *6* 3, 1925

Albers Schonberg Zur Bestimmung der Herzgrosse mit besonderer Berucksichtigung der Orthophotographie (Distanzaufnahme, Telerontgenographie) Fortschr a d. Geb d Rontgenstrahlen, *12* 38, 1908

Albert, A H, and Klaus, E J See Klaus

Albrecht, H U Zur Rontgendiagnostik der Aneurysmen der Sinus Valsalvae der Aorta Fortschr a d Geb d Rontgenstrahlen, *53* 218, 1936

Altschul, [illegible]

Altstaedt [illegible]

Alwens, [illegible]
klin [illegible]

Amelung, W Die Veranderungen des Rontgenbildes der Brustorgane bei Kyphoskoliosen und Skoliosen Fortschr a d Geb d Rontgenstrahlen, *28* 230, 1921/22.

Amelung, W Zur Frage der doppelten Konturierung des Herzschattens im Rontgenbilde bei Pericarditis Fortschr a d. Geb d Rontgenstrahlen, *28* 519, 1921/22.

Ameuille, P, and Salles, P Sur le diagnostic radiologique de la péricardite tuberculeuse Bull. Soc. méd d hôp de Paris, *48* 200, 1932

Hultén). In certain projections one sees, instead of the drop-like cross-section of the azygos vein, a loop projecting from the mediastinal shadow in the infraclavicular part of the lung field, corresponding to the entire course of the intralobar section of the vena azygos (Zdansky) (fig. 334a and b).

In the left anterior oblique position, very often the shadow of the azygos vein joins the superior vena cava and the interlobar fissure of the azygos lobe (Zdansky) (fig 333b)

Awareness of the lobus vena azygos is important if confusion of this common finding with intrapulmonary fibrosis or pleural strands is to be avoided. Moreover, through it some atypical extensions of pathologic lung processes become comprehensible, tuberculous and nonspecific consolidations can involve the accessory lobe alone or spare it completely. By thickening of the interlobar pleura the interlobar strip may become broader and irregularly outlined and the accessory lobe more or less widely obscured (Hippe and Huhle).

B

Bahn, K.. Ueber isolierte Dextrocardie mit Isthmusstenose der Aorta und Endocarditis lenta Dtsch Arch. f klin Med, *146* 297, 1925.

Bainbridge, F. A · The Physiology of Muscular Exercise London, Longmans, Green & Co, 1931

Baird, L W, and Kirklin, B. R. Obstruction of the third portion of the duodenum of unusual etiology, report of case Radiology, *27* 235, 1936

Baker, C, and Trounce, J. R.· Arteriovenous aneurysm of the lung Brit Heart J, *11* 109, 1950.

Baker, T W, and Shelden, W. D. Coarctation of the aorta with intermittent leakage of a congenital cerebral aneurysm, report of a case Am J M Sc, *191* 626, 1936.

Ball, D · Change in size of the heart in severe anemia Am. Heart J, *6* 517, 1931.

Ball, R. P. Cor biatria triloculare with transposition of arteries Am J Dis Child, *32* 84, 1923

Bamberg, K, and Putzig, H.· Die Herzgrosse im Sauglingsalter auf Grund von Rontgenfernaufnahmen Ztschr. f Kinderheilk, *20* 195, 1919

Baranova, A : Ueber die Rontgendiagnose der Atherosklerose der Brustschlagader Rontgenpraxis, 7:175, 1935.

Barcroft, J Die Atmungsfunktion des Blutes. I. Teil Berlin, J Springer, 1927

Bardeen, C. R Tables for aid in the determination of the relative size of the heart by means of roentgen-rays Am. J. Roentgenol, *4* 604, 1917

Bardeen, C R. Determination of the size of the heart by means of x-rays Am J Anat, *23* 423, 1918

Barnes, J M, and Stedem, D E. Multiple aneurysms of the smaller branches of the pulmonary artery Am J Roentgenol, *30* 443, 1933

Barret, G Localisation radiologique d'un projectile intracardiaque libre et mobile dans le ventricule droit, J Radiol. (Belg), *2*:37, 1916

Barsony, T, and Koppenstein, E · Lobus apicodorsalis (eine bisher unbekannte Lokalisation des Azygoslappens) Fortschr a d Geb d Rontgenstrahlen, *41* 459, 1930

Bastenie, P Zur Frage des Basedow-Herzens Beitr z path Anat u z allg Path, *94* 361, 1934

Bates, J C, and Leaver, F Y Pericardial coelomic cysts Presentation of five cases and five similar cases illustrating difficulty of diagnosis Radiology, *57* 330, 1951

Bauer, J, and Helm, J. Ueber Rontgenbefunde bei Kropfherzen Dtsch Arch f klin. Med, *109* 72, 1913

Bauke, E E Ueber Rontgenbefund bei einem perforationsreifen Herzspitzenaneurysma Rontgenpraxis, *5* 444, 1933

Baumann, W, and Naumann, W Rontgenanatomische Befunde am Herzskelett beim totalen Herzblock. Fortschr a d. Geb. d Rontgenstrahlen, *56* 723, 1937

Baumeyer, S Ueber ein mehrteiliges nicht luetisches Aortenaneurysma Dtsch med Wchnschr, *62* 1369, 1936

Baumgartner, E. A, and Abbott, M. E Interventricular septal defect with dextroposition of

111 639, 1929

Bayley, R H Dynamic dilatation of the thoracic aorta Am Heart J, *8* 585, 1933

Becher, E Zur Casuistik der Herzsteckschusse Munchen med Wchnschr, *65* 429, 1918

Becker, T. Die Analyse des Elektrokardiogramms mittels der Rontgenkymographie Dtsch Arch f klin. Med, *113* 216, 1914

Becker, W Ueber Periarteritis nodosa Med Klin, *34* 869, 1938

Bedford, D E Two cases of aneurysmal dilatation of the left auricle Proc Roy Soc Med, *20* 10, 1927

Bedford, D E, and Parkinson, J · Right sided aortic arch (Situs inversus arcus aorta) Brit. J Radiol, *9* 776, 1936

Bellet, S, amd Stewart, H L Congenital heart disease—atresia of the tricuspid orifice Am J Roentgenol, *45* 1247, 1933

Bendick, A J, and Wessler, H Azygos lobe of the lung Am. J Roentgenol, *20* 1, 1920

Andersson, T. Electrokymographic studies in pulmonary stenosis and tetralogy of Fallot Acta rad., *36* 345, 1951

Antenucci, A J Ueber Pulmonalsklerose Wien Arch f. inn. Med, *26* 197, 459, 1935

Anthony, A J, and Koch, A Das Herzminutenvolumen des Gesunden bei weiderholter Bestimmung mit verschiedenen Methoden. Dtsch Arch. f klin Med., *177* 158, 1934.

Antopol, W., Heilbrunn, J, and Tuchman, L. Enlargement of the heart due to abnormal glycogen storage Am J M Sc, *188* 354, 1934

Arendt, J Radiological differentiation between pericardial effusion and cardiac dilatation Radiology, *50* 44, 1948

Arens, R A., and Stewart, E Pneumocardium following a foreign body in esophagus Radiology, *22* 234, 1934

Arkin, A Totale Persistenz des rechten Aortenbogens im Rontgenbild Wien Arch f inn Med, *12* 385, 1926.

Arkin, A Double aortic arch with total persistence of the right and isthmus stenosis of the left arch a new clinical and X-ray picture Am Heart J, *11* 444, 1936

Arkussky, J Zur Frage der Rontgendiagnostik der angeborenen Herzfehler, basiert auf 23 Falle, von denen zwei durch Sektion bestatigt Fortschr. a d Geb d Röntgenstrahlen, *35* 455, 1926.

Arkussky, J. Das Rontgenbild der angeborenen Stenose der Lungenarterie bei gleichzeitiger Existenz eines Defekts der Kammerscheidewand Fortsch a d. Geb d Rontgenstrahlen, *41* 617, 1930

Arkussky, J Zur rontgenologischen Charakteristik der Funktionsfähigkeit des Herzens. Fortschr. a d Geb d Rontgenstrahlen, *41* 714, 1930.

Arkussky, J Neue Ergebnisse zur Frage der Orthodiagraphie des Herzens Fortschr a d Geb d Rontgenstrahlen, *44* 39, 1931

Arnheim, G Ein Fall von angeborener Pulmonalstenose sowie Bemerkungen uber die Diagnose des offenen Ductus Botalli Berl klin Wchnschr., *42* 106, 1905

Arnstein, A Eine seltene Missbildung der Trikuspidalklappe Arch f path Anat., *266* 247, 1927

Arrillaga, F C Anévrisme de l'artére pulmonaire chez les cardiaque noirs Bull d Soc méd d hôp. de Paris, *48* 1230, 1924

Arrillaga, F. C., Donovan, C, and Taquini, A C Diverticulo del pericardio. Rev. argent de cardiol, *5* 49, 1938

Aschenbrenner, R Operative Behandlung schwerer Herz- und Kreislaufdekompensation. Beitrag zur Klinik der Spatfolgen arteriovenoser Aneurysmen Klin Wchnschr, *13* 689, 1934

Ascoli, V Migration d'un projectile a travers la veine cave inférieure jusque dans l'oreillette droit Arch d mal du coeur, *10* 283, 1917.

Ashworth, H, and Jones, A M Aneurysmal dilatation of the left auricle with erosion of the spine Brit Heart J, *8* 207, 1940.

Assmann, H Das Myxodemherz, Munchen. med Wchnschr., *66* 9, 1919

Assmann, H Ueber Veranderungen des Hilusschattens bei Herzkrankheiten Munchen med Wchnschr, *67* 177, 1920.

Assmann, H. Die klinische Rontgendiagnostik der inneren Erkrankheiten 5 Aufl, Berlin, F C W Vogel, 1934

Atlas, D H, Eisenberg, H, and Gaberman P Bernheim's syndrome Circulation *1* 753, 1950.

Attinger, E. Die Interpretation des hinteren Herzrandes in frontaler und schrager Durchleuchtung. Fortschr a d Geb d Rontgenstrahlen, *31* 62, 1923.

Attinger, E. Ueber die Beobachtung eines Falles von paroxysmalem Herzkammerflimmern. Zentralbl f. Herz u Gefass Krankh, *16* 245, 1924

Aubertin, C, and Horeau, J Anévrisme du ventricule gauche apparu six semaines apres un infarctus du myocarde, diagnostic par la radioscopie et le radiokymographie Bull d l Soc. méd. d hôp. de Paris, *54* 730, 1938

Averbuck, S H.: Heart failure in hypertension Am Heart J, *11* 99, 1936

Axén, O., and Lind, J.: Table for routine angiocardiography, synchronous serial roentgenography in two planes at right angles. J A M A., *143* 540, 1950

Ayman, J., Rosenblum, H, and Falcon-Lesses, M "Myxedema heart" without evidence of cardiac insufficiency J A M A, *98* 172, 1932.

B

Bahn, K · Ueber isolierte Dextrocardie mit Isthmusstenose der Aorta und Endocarditis lenta Dtsch Arch. f klin. Med , *146* 297, 1925

Bainbridge, F. A The Physiology of Muscular Exercise London, Longmans, Green & Co , 1931

Baird, L W, and Kirklin, B R Obstruction of the third portion of the duodenum of unusual etiology, report of case Radiology, *27* 235, 1936

Baker, C., and Trounce, J. R Arteriovenous aneurysm of the lung Brit Heart J , *11* 109, 1950

Baker, T W., and Shelden, W. D Coarctation of the aorta with intermittent leakage of a congenital cerebral aneurysm, report of a case. Am. J M Sc., *191* 626, 1936

Ball, D Change in size of the heart in severe anemia Am. Heart J , *6* 517, 1931

Ball, R P · Cor biatria triloculare with transposition of arteries Am J Dis Child , *32* 84, 1923

Bamberg, K , and Putzig, H. Die Herzgrösse im Sauglingsalter auf Grund von Rontgenfernaufnahmen. Ztschr. f. Kinderheilk, *20* 195, 1919

Baranova, A : Ueber die Röntgendiagnose der Atherosklerose der Brustschlagader. Rontgenpraxis, 7 175, 1935.

Barcroft, J : Die Atmungsfunktion des Blutes. I. Teil Berlin, J Springer, 1927

Bardeen, C. R. Tables for aid in the determination of the relative size of the heart by means of roentgen-rays. Am J Roentgenol., *4* 604, 1917

Bardeen, C. R.: Determination of the size of the heart by means of x-rays Am J Anat , *23* 423, 1918.

Barnes, J M , and Stedem, D E. Multiple aneurysms of the smaller branches of the pulmonary artery Am J Roentgenol , *30* 443, 1933

Barret, G Localisation radiologique d'un projectile intracardiaque libre et mobile dans le ventricule droit, J Radiol (Belg), 2.37, 1916

Barsony, T, and Koppenstein, E.· Lobus apicodorsalis (eine bisher unbekannte Lokalisation des Azygoslappens). Fortschr. a d Geb d. Röntgenstrahlen, *41* 459, 1930

Bastenie, P. Zur Frage des Basedow-Herzens Beitr z path Anat u z allg Path , *94* 361, 1934

Bates, J C , and Leaver, F Y Pericardial coelomic cysts Presentation of five cases and five similar cases illustrating difficulty of diagnosis Radiology, *57* 330, 1951

Bauer, J , and Helm, J.· Ueber Röntgenbefunde bei Kropfherzen Dtsch Arch f klin. Med , *109* 72, 1913.

Bauke, E E · Ueber Rontgenbefund bei einem perforationsreifen Herzspitzenaneurysma Rontgenpraxis, *5* 444, 1933.

Baumann, W, and Naumann, W Rontgenanatomische Befunde am Herzskelett beim totalen Herzblock Fortschr. a d. Geb d. Rontgenstrahlen, *56* 723, 1937

Baumeyer, S Ueber ein mehrteiliges nicht luetisches Aortenaneurysma Dtsch med Wchnschr , *62* 1369, 1936.

Baumgartner, E A., and Abbott, M E.. Interventricular septal defect with dextroposition of aorta and dilatation of the pulmonary artery ("Eisenmenger complex") terminating by cerebral abscess. Report of a case observed during life, presenting impaired conduction and paralysis of recurrent laryngeal nerve from pressure of hypertrophied pulmonary conus Am J M Sc , *177* 639, 1929.

Bayley, R. H.: *Dynamic dilatation of the thoracic aorta Am. Heart J , 8 585, 1933*

Becher, E. Zur Casuistik der Herzsteckschusse. Munchen med Wchnschr., *65* 429, 1918

Becker, T : Die Analyse des Elektrokardiogramms mittels der Rontgenkymographie. Dtsch Arch f klin. Med , *113* 216, 1914

Becker, W. Ueber Periarteritis nodosa Med Klin , *34* 869, 1938.

Bedford, D E.· Two cases of aneurysmal dilatation of the left auricle Proc. Roy Soc. Med , *20* 10, 1927.

Bedford, D E., and Parkinson, J.: Right sided aortic arch (Situs inversus arcus aortae). Brit J Radiol , *9* 776, 1936

Bellet, S , amd Stewart, H L · Congenital heart disease—atresia of the tricuspid orifice Am J Roentgenol , *45* 1247, 1933.

Bendick, A. J., and Wessler, H : Azygos lobe of the lung Am. J Roentgenol , *20* 1, 1920

Benedetti, P. Die klinische Morphologie des Herzens und ihre Auswertungsmethodik bei Herzgesunden und Herzkranken. Ergebn inn Med, *51*:531, 1936
Benhamou, E Echinokokkuszyste des Herzens. Arch mal d coeur., *31* 137, 1938
Benjamin, J E, and Wiedemer, L A case of aortic insufficiency *due to rupture of aortic valve.* Med. Bull. Univ Cincinnati, *2* 113, 1933.
Berger, W, and Olloz, M Untersuchungen zum Studium des Trainiertseins III. Mitt Herzgrösse und Herzschlagvolum Arb physiol, *7* 1933, cited after Zbl Radiol., *16*, 1934.
Bergk, K, and Chantraine, H Ueber eine bequeme und zuverlassige Vorrichtung zur Einschaltung der Lungenaufnahmen durch den Herzschlag. Fortschr a d Geb d Rontgenstrahlen, *45*:334, 1932
v Bergmann, G. Das "epiphrenale Syndrom," seine Beziehung zur Angina Pectoris und zum Kardiospasmus, Dtsch med Wchnschr., *58* 605, 1932.
Berk, L. H. Roentgen diagnosis of mural thrombi, Arch Int. Med, *63* 1183, 1939.
Berner, F Ein neues Rontgensymptom bei Herzbeutelerguss Fortschr a d Geb d Rontgenstrahlen, *56* 53, 1937.
Berner, F Die Differentialdiagnose zwischen Accretio und Concretio pericardii circumscripta im Kymogramm Dtsch Arch f klin Med, *182* 88, 1938.
v Bernuth, F. Radiologische Untersuchungen uber die Herzgrosse im Kindesalter Ergebn inn Med, *39* 69, 1931
Besser, F, and Schilling, C Zur Klinik und Rontgenologie der Herzthromben Dtsch. Arch f klin Med, *175* 50, 1933
Bickenbach, O Die Messung des Querschnitts der Aorta descendens usw. Dtsch Arch f Klin. Med, *171*,647, *1931*
Bier, A Hand d arztl Erfahrung im Weltkrieg Bd II
Binhold, H Das Herzvolumen in der Schwangerschaft. Arch f Gynak, *154* 25, 1933.
Binhold, H Ueber das Herzvolumen bei Emphysem, Asthma und Tuberkulose der Lunge Ztschr f Kreislaufforsch, *27* 14, 1935
Bischoff, S. Zum klinische Bild der Glykogenspeicherungskrankheit Ztschr f Kinderheilk, *52* 722, 1932
Bishop, L F, Kirschner, P, and Pessar, T. Diverticulum of the pericardium Circulation, *1* 813, 1950
Bishop, P A, and Roesler, H The roentgenologic diagnosis of intracardiac calcifications Am J Roentgenol, *31* 1, 1934
Bittorf, A, and Hubner, L Der Oesophagus bei Kyphoscoliosen im Rontgenbilde Fortschr a d Geb. d Rontgenstrahlen, *33* 59, 1925
Blackford, L M Coarctation of the aorta Arch Int Med, *41* 702, 1928
Blalock, A The surgical treatment of congenital pulmonic stenosis. Ann Surg *124* 879, *1946.*
Blalock, A., and Taussig, H B The surgical treatment of malformations of the heart in which there is pulmonary stenosis or pulmonary atresia J A M A, *128* 189, 1945
Blauel, Muller, O, and Schlayer Ueber das Verhalten des Herzens bei Struma Beitr z klin Chir., *62* 119, 1909
Block, W J, Parker, R L, and Edwards, J. E "Myxoma" of the left atrium clinically simulating mitral stenosis Report of case and pathologic studies Proc Staff Meetings, Mayo Clinic *27* 361, 1952
Blumberger, K Die Untersuchung d Dynamik des Herzen beim Menschen Ihre Anwendung als Leistungprufung. Ergebn. inn Med, *62* 149, 1942
Blumenfeldt E Die angeborenen Herz- und Gefasskrankheiten In Kraus-Brugsch Spezielle Pathologie und Therapie innerer Krankheiten Bd 4, S 449
[illegible] fémorale par
[illegible] Geb d Ront-
[illegible] Funktion als
Saugpumpe wahrend der Systole Klin. Wchnschr, *14* 614, 1935.
Bohme, W · Ueber zwei neue, von der Respiration abhangige, klinisch erfassbare, rontgenologisch geklarte Herzphanomene. Fortschr a d. Geb. d. Rontgenstrahlen, *55* 107, 1937

Bohnenkamp, H Lehrbuch d spez physiol Pathologie fur Studierende u Aerzte Jena, 1935
Boller, R, and Paper, R Zur Diagnose der Herzaneurysmen Fortschr a d Geb d Röntgenstrahlen, *45* 318, 1932.
Bollini, V. Note di cardiovolumetria sperimentale Radiol e Fisica med, *2* 193, 258, 1935
Boone, R. B, Chamberlain, W E, Gillick, F. G, Henny, G C, and Oppenheimer, M J Interpreting the electrokymogram of heart and great vessels Am Heart J, *34* 560, 1947
Borak, J Zwerchfellveranderungen bei Coronarverschluss. Wien klin Wchnschr, *49* 939, 1936
Bordet, E, and Fischgold, H. Verification de la loi de Starling par la radiologie du coeur Arch d mal. du coeur, *28* 198, 1935
Bordet, E, and Lereboullet, J Gros anévrisme de l'origine de l'aorte simulant une écrasie de l'artère pulmonaire Arch d mal du coeur, *22* 314, 1929
Böttner, A. Ueber die Diagnose der Aneurysmen der Aorta abdominalis mit besonderer Berucksichtigung der direkten Röntgendiagnostik Munchen med Wchnschr, *66* 296 1911
Boyd, L J, and McGavack, T H Aneurysm of the pulmonary artery review of literature and report of two new cases Am Heart J, *18* 562, 1929
Bradley, E B, and Maxwell, E S Primary neoplasms of the heart J A M A, *91* 1352, 1928
Brahms, A W, and Strauss, A H The effect of amyl nitrite on the size of the heart and the width of the aortic shadow as determined roentgenologically Am J M Sc, *180* 618, 1930
Brailsford, J F Aneurysm of the abdominal aorta diagnosis by lateral radiography of the spine Brit J Surg, *14* 369, 1926
Brailsford, J F Pneumopyopericardium the radiological diagnosis, Brit M J, *2* 1053, 1929
Bramwell, C Coarctation of the aorta Brit Heart J, *9* 100, 1947
Bramwell, J C, and Duguid, J B Aneurysmal dilatation of the left auricle Quart J Med, *21* 187, 1928
Bramwell, J C, and Ellis, R Some observations on the circulatory mechanism in marathon runners Quart J Med, *24* 329, 1930/31
Braun, K, de Vries, A, Feingold, D S, Ehrenfeld, N E, Feldman, J, and Schorr, S Complete dextroposition of the aorta, pulmonary stenosis, intraauricular septal defect and patent foramen ovale Am Heart J, *43* 773, 1952
v Braunbehrens Die Hermuskelschwiele und das Herzwandaneurysma Fortschr a d Geb d Rontgenstrahlen, *50* 15, 1934
Brednow, W Plastische Darstellung des Herzens Ztschr f klin Med, *122* 382, 1932
Brednow, W. Polycythaemia vera im Rontgenbild, zugleich ein Beitrag zur rontgenologischen Darstellung der Stauungslunge Rontgenpraxis, *5* 732, 1933
Brednow, W, and Schaare, U Kymographische Untersuchungen des normalen Herzens Ztschr f klin Med, *125* 480, 1933
Bredt, H Die Missbildungen des menschlichen Herzens Ergebn allg Path u path Anat, *30* 77, 1936
Breitmann, M Eine einfache Formel zur Bestimmung des transversalen Durchmessers des Herzens nach Korpergrösse, Korpergewicht und Brustumfang, Ztschr f Kreislaufforschg, *23* 767, 1931
Brenner, F, and Wachner, S Ueber einen ungewohnlichen Sitz eines Herzaneurysmas und seine Rontgendiagnostik Fortschr a d Geb d Rontgenstrahlen, *54* 243, 1936
Brill, J C, and Robertson, T J Subacute cor pulmonale Arch Int Med, *60* 1043, 1937
Broden, B, Jonsson, G, and Karnell, J Thoracic aortography Observations on technical problems connected with the method and various risks in its use Acta rad, *32* 498, 1949, Thoracic aortography in the diagnosis of patent ductus arteriosus Acta rad, *34* 65, 1950
Brown S E and Giffin, H Z Studies of the vascular changes in cases of polycythemia vera Am J M Sc, *171* 157, 1926
Brugsch, T Ueber das Verhalten des Herzens bei Skoliose Munchen med Wchnschr., *57* 1741, 1910
Brunetti, L Aorta alta destra e disfagia luscoria Riv Radiol e Fisica med, *5* 76, 1931.
Bruns, O Herzgrosse und Muskelarbeit Ergebn Inn Med u. Kinderh, *34* 201, 1928
Bruns O, and Roemer, G A Der Einfluss angestrengter körperlicher Arbeit auf radiographische Herzgrosse Blutdruck und Puls Ztschr f klin Med, *94* 22, 1922.

v Buchem, and Polak, D Elektrokardiographische Untersuchungen bei akuten Infektionskrankheiten Dtsch Arch f klin Med., *174* 250, 1933.

Burchell, H B. Variations in clinical and pathological picture of patent ductus arteriosus M. Clin North America, *32* 911, 1948.

Burger, M. Ueber die Bedeutung des intrapulmonales Drucks fur den Kreislauf und den Mechanismus des Kollapses bei akuten Anstrengungen Klin Wchnschr, *5* 777, 825, 1926

Burger, M, and Petersen, P. F Die Pressdruckprobe als Herzleistungsprufung, Arbeitsphysiologie, 1929, 1

Burger, M., and Petersen, P F. Rontgenologische Herzfunktionsprufung Fortschr a d Geb d. Rontgenstrahlen, *60* 78, 1939.

Burke, E C, Kirklin, J. W, and Edwards, J E Sites of obstruction to pulmonary blood flow in the tetralogy of Fallot, an anatomic study Proc Staff Meetings Mayo Clin, *26* 498, 1951.

Burke, J Ueber angeborene Pulmonalstenose Ztschr. f Heilk., *23* 56, 1902.

Burke, J Ueber angeborenene Enge des Aortensystems. Dtsch. Arch f klin Med, *71* 189, 1901.

Butler, P F, and Dana, H W Diaphragm excursions, normal and pathologic. Am J M Sc, *176* 569, 1928.

C

Cabrera, E C, and Monroy, J R Sites of obstruction to pulmonary blood flow in the tetralogy of Fallot, an anatomic study Am Heart J, *43* 661, 669, 1952.

de la Camp Experimentelle Studien uber die akute Herzdilatation. Ztschr f klin Med, *51* 1, 1904.

de la Camp. Beitrage zur Physiologie und Pathologie der Zwerchfellatmung einschliesslich der zugehorigen Herzbewegungen Ztschr f klin Med, *49* 411, 1903.

Campbell, M Dissecting aneurysm with survival for three months after rupture into the pleura Brit. Heart J, *8* 200, 1946

Campbell, M., and Gardner, F. Radiological features of enlarged bronchial arteries Brit Heart J, *12* 184, 1950.

Campbell, M., Gardner, F., and Reynolds, G Cor biloculare Brit Heart J, *14*·317, 1952.

Campbell, M, and Hills, T H Angiocardiography in cyanotic congenital heart disease Brit Heart J., *12* 65, 1950

Campbell, M, and Sussman, S Coarctation of the aorta Brit Heart J, *9* 304, 1947.

Canigiani, T Zur Rontgendiagnostik der Aneursyma dissecans der Brustaorta Fortschr. a d Geb d Rontgenstrahlen, *45* 416, 1932.

de Carvalho, L, Moniz, E, and Saldanha, A. La visibilité des vaisseaux pulmonaires J. Radiol, *16* 469, 1932.

Castellanos, A, Pereiras, R, and Garcia, O Angiocardiography Anatomo-roentgenographical forms of the transposition of the vessels Am J Roentgenol., *64* 255, 1950

Castex, M R., di Ciou, A V, and Battros, A Anevrisme de la branche droite de l'artère pulmonaire. Arch méd-chir appar. respirat., *6* 1, 1931

Chamberlain, W. E, and Dock, W The study of the heart action with roentgen cinematograph Radiology, *7* 185, 1926

Chantraine, H, and Bergk, K : Ueber eine bequeme und zuverlassige Vorrichtung zur Einschaltung der Lungenaufnahmen durch den Herzschlag. Fortschr. a d Geb d Rontgenstrahlen *45* 334, 1932.

Chaperon, R.· Étude radiologique du déroulement aortique Bull acad méd Paris, *41*.355, 1927.

Chaoul, H, and Adam, A Die Hiatushernie im Rontgenbild und ihre Abrenzung gegenuber funktionelle Zustanden des unteren Abschnittes des Oesophagus Fortschr a d. Geb d. Rontgenstrahlen, *46* 143, 1932

Chiari, H · Ueber Veranderungen in der Adventitia der Aorta und ihren Hauptasten im Gefolge von Rheumatismus Beitr z path Anat u z allg Path, *80* 336, 1928.

Chiari, H Ueber Myokarditis Wien klin Wchnschr, *46* 1137, 1933

[illegible] der patholog Anatomen Wiens u. der [illegible] Gemeinschaftl Sitzung der patholog Anatomen Wiens u der Ges f. innere Medizin in Wien, 22, IV, 1937

Christeller, E Funktionelles und Anatomisches bei der angeborenen Verengerung und dem angeborenen Verschluss der Lungenarterie, insbesondere über die arteriellen Kollateralbahnen bei diesen Zustanden Arch. f path Anat , *223* 399, 1916/17

Christian and Frik, K.. Rontgenbefund bei chronischen partiellem Herzaneurysma Klin Wchnschr , *1* 582, 1922.

Cignolini, P. Tabelle delle dimensioni normali radiogiche del fascio cardiovascolare e dei polmoni nei maschi fra 1 6 ed in 17 anni Atti ufficiali del II Convengo dei Medicini dell'O N B

Cignolini, P. Röntgenchimografica Cardiaca e Regmografia Bologna, Capelli, 1934

Cignolini, P. Die Röntgenkymographie mit unterbrochenem Schlitz Fortschr. a Geb d. Rontgenstrahlen. *49* 224, 1934

Clark, A La survie en cas d'anévrisme pariétal du coeur Arch d mal du coeur *34* 143, 1941

Clark, J J Calcification of the pericardium and within the heart muscle Radiology *35* 356, 1940

Claytor, T A , and Merrill, W H Orthodiagraphy in the study of the heart and great vessels. Am J M Sc , *138* 549, 1909

Clerc, A., Bascouret, M , and Froyer, M Communication entra l'aorte et l'artère pulmonaire par rupture d'anévrisme, avec survie de plus de quatre ans Bull d Soc méd d hôp de Paris *55* 128, 1933

Clerc, A., Nöel Deschamps, P , and Schwob, R Volumineux anévrisme disséquant aortique ayant simulé un néoplasme pulmonaire. Bull. d Soc Méd d. hôp. de Paris, *47* 280, 1931

Coe, F O , and Otell, L E Acute pulmonary edema Am J Roentgenol, *27* 101, 1932

Cooley, R N , and Sloan, R D Angiocardiography in congenital heart disease of cyanotic type III Observations on complete transposition of the great vessels. Radiology, *58* 481, 1952.

Cooley, R. N , Sloan, R D , Hanlon, C R , and Bahnson, D T Angiocardiography in congenital heart disease of cyanotic type II Observations on tricuspid stenosis or atresia with hypoplasia of the right ventricle Radiology, *54* 848, 1950

Conrads, B Ueber die durch gesunde und krankhaft veranderte Nachbarorgane bedingt Eindellungen der Speiserohre und ihr Nachweis im Rontgenbild Rontgenpraxis, *9* 750, 1937

Conta, G. Periarteritis nodosa der Lungengefasse u Lungenröntgenbild Fortschr a d Geb d Rötngenstrahlen, *47* 510, 1933

Conte, E., and Costa, A Angiopneumography Radiology, *21* 465, 1933

Cope, Z Extraction of a sewing needle from the heart Lancet, *1* 813, 1920

Costa, A Morfologia e patogenesi degli aneurismi dell' arteria polmonare (sopra un caso di voluminosi aneurismi multipli de tronco e dei grossi e medi rami, su base malformativo) Arch pat e. clin med , *8* 257, 1929

Cottenot, P. Sélecteur cardio-respiratoire J Radiol et Électrol , *17* 381, 1933

Cottenot, P L'image radiologique du coeur en systole et en diastole Bull. et Mém Soc rad méd de France, *21* 52, 1933

Cottenot, P , and Heim du Balsac, R Étude de kymographie cardiovasculaire, Ann de méd , *39* 24, 1936

Cournand, A Cardiac catheterization New York, 1949 Commonwealth Fund

Cowan, J A , Harrington, A W, and Riddell, J R On pneumopericardium Quart J Med , *7* 165, 1914

Crafoord, C , and Nylin, G Congenital coarctation of the aorta and its surgical treatment J. Thoracic Surg , *14* 347, 1945

Cramer, H , and Stehr, L Ergebnisse der Kymographie bei Herzbeutelaffektionen Fortschr a d. Geb d Rontgenstrahlen, *56* 404, 1937

Crane, A W Roentgenology of the heart Am J. Roentgenol *3* 512, 1916

Crane, P H , Lerner, H H , and Lawrence, E A The syndrome of arteriovenous fistula of the lung Am J Roentgenol , *62* 418, 1949

Crawford, J H , and de Veer, J A, Aneurysm of the aorta producing pulmonary stenosis and bundle branch block Am Heart J , *7* 789, 1932

Crowden, S P , and Harris, H A The effect of obstructed respiration on heart and lungs Brit. M J , *1* 439, 1929.

Curschmann H Zur Beurteilung und operativen Behandlung grosser Herzbeutelergusse Dtsch. Klin , 1907, 4

Curschmann, H Emphysem und Herz Med Klin, *24* 921, 1928.

Cutler, E C, and Sosman, M C. Calcification of the heart and pericardium. Am J Roentgenol, *12* 312, 1924

Czepa, A Zur Differentialdiagnose zwischen Lungentumoren und Aneurysma Fortschr a d Geb. d *Rontgenstrahlen*, *29* 277, 1922

D

Dack, S, Sussmann, M L, and Master, A. M The roentgenkymogram in myocardial infarction. 2 Clinical and electrocardiographic correlation. Am Heart J, *19* 464, 1940

Dahm, M Die *Bewegungen des Oesophagus im Rontgenbild Fortschr a d Geb. d Rontgenstrahlen*, *43* 464, 1921

Danelius, G Experimentelles uber den Verlauf der oberen Lungengrenze im Rontgenbilde Fortschr a d Geb d Rontgenstrahlen, *40* 249, 1929

Danelius, G. Absence of the hilar shadow, a diagnostic sign in rare congenital malformations (truncus arteriosus solitarius) with heterotopic pulmonary blood supply. Am J Roentgenol, *47* 890, 1952

Danzer, C S. *The cardiothoracic ratio An index of cardiac enlargement. Am J M. Sc*, *157* 513, 1919

Darley, W, and Doan, C A Primary pulmonary arteriosclerosis with polycythemia assocated with the chronic ingestion of abnormally large quantities of sodium chloride (Halophagia) Am J. M. Sc, *191* 633, 1936

Dautrabande, L Blood output by heart in anemia Compt rend. d. Soc de Biol, *93* 1029, 1925

Davis, D, Weinstein, A A, Riseman, and Blumgart, H L. Treatment of chronic heart disease *by total ablation of the thyroid gland VII The heart in artificial myxedema* Am Heart J, *10* 17, 1934

Debeyre, A, and Lorgnier Un cas de migration tardive d'une balle de fusil de la veine cave inférieure au ventricule droit. J Radiol, *3* 66, 1918

Dedic, S Das mitralstenotische und das mitralkonfigurierte Herz im Profil Fortschr. a d Geb d Rontgenstrahlen, *38* 68, 1928.

Dedic, S Die proportionelle Aortenmessung in der Rontgenologie. Fortschr. a d Geb d *Rontgenstrahlen*, *50* 42, 1934

Dedichen, L Norsk med Laeger, *5* 1920. Cited by Reindell.

Delherm, L, Bordet, E, Thoyer-Rozat, P, and Fischgold, H Étude sur les courbes radiokymographiques de la révolution cardiaque. Arch d mal du. coeur, *26* 653, 1933.

Delherm, L, and Fischgold, H Quatre années de radiokymographie cardiovasculaire Fortschr a d Geb Rontgenstrahlen, *53* 223, 1936

Delherm, L, Thoyer-Rozat, P, and Fischgold, H. Sur le détermination du point G et sur la *radiographie du coeur en systole et en diastole* Bull d l Soc d méd d hôp de Paris, *21* 45, 1933

Dell'Acqua, S, and Freundlich, J Ueber sackformige Abkapselung eines perikarditischen Exsudates Ztschr f Kreislaufforsch, *20* 703, 1929

Deneke, T Die Aorta im Rontgenbilde Dtsch med Wchnschr., *50* 293, 1924.

Deneke, T Die pathologische Aorta im *Rontgenbilde. Fortschr. a d. Geb d Rontgenstrahlen*, *33* (Kongressheft), 1925

Deneke, T Zur Klinik der Isthmusstenose der Aorta Arch f path. Anat, *254*, 336, 1925.

Deterling, R A, and Clagett, O T. Aneurysm of the pulmonary artery, review of literature and report of case Am Heart J, *34* 471, 1947.

Determann, A Beitrag zur Differentialdiagnose der Verschattungen in der *Herzsilhouette* Fortschr a d Geb d Rontgenstrahlen, *46* 137, 1932

Determann, A Ueber das *Verhalten des Gefassbandes*, inbesondere der Aorta bei der Atmung [illegible] 1937.

[illegible] and Wenger, R Die Elektrokymographie.

[illegible] 632, 670, 1928

[illegible] schr, *46* 849, 1933.

[illegible] amt Wien, 1934

Deutsch, F., and Kauf, F Herz und Sport, Berlin, 1924
Diehl, F, and Kuhlmann, F Die Knochenbildungen in der Lunge mit besonderer Berucksichtigung der tuberösen Form. Fortschr a d Geb. d Rontgenstrahlen, *48* 202, 1933
Dietlen, H Orthodiagraphische Beobachtungen uber Veranderungen der Herzgrosse bei Infektionskrankheiten, bei exsudativer Perikarditis und paroxysmale Tachykardie, nebst Bemerkungen uber das röntgenologische Verhalten der Pneumonie, Munchen med Wchnschr, *50* 2077, 1908
Dietlen, H Klinische Bedeutung der Veranderungen am Zirkulationsapparate inbesondere der wechselnden Herzgrosse, bei verschiedenen Korperstellungen (Liegen und Stehen), Dtsch Arch f klin Med, *97*·132, 1909.
Dietlen, H · Zur Frage des "kleinen Herzens," Munchen med. Wchnschr, *66* 9, 47, 1919
Dietlen, H · Ueber die Unterscheidung von Hypertrophie und Dilatation im Rontgenbild, Zentralbl. f Herz u Gefasskrankh, *13*,315, 1921
Dietlen, H Ueber Herzgrosse und Herzmessung, Klin Wchnschr, *1* 2097, 1922.
Dietlen, H Herz und Gefasse im Rontgenbild, Leipzig, Barth, 1923
Dietlen, H. Zur Frage der akuten Herzerweiterung bei Kreigsteilsnehmern Munchen med. Wchschr, *58* 182, 1916
Dietlen, H Das Rontgenbild der Mitralstenose. Ztschr. f Kreislaufforsch, *20* 697, 1928
Dietlen, H Probleme der Pathologie des Kreislaufes Dtsch med Wchnschr., *54* 2006, 1928
Dietlen, H Cor bovinum oder Herzbeutelerguss? Wien. Arch f inn Med, *18* 19, 1929
Dietlen, H Fortschritte in der Rontgendiagnostik Munchen. med Wchnschr, *82* *108*, *1935*.
Dietlen, H Herztonus Handb d norm u path Phys, Bd VII/I, p 364
Dietlen, H, and Moritz, F Ueber das Verhalten des Herzens nach langedauernden und anstrengendem Radfahren Munchen med Wchnschr, *55* 489, 1908
Dietlen, H, and Schall, L Die Rontgendiagnostik des kindlichen Herzens Handbuch der Rontgendiagnostik und Therapie im Kindesalter, Leipzig, Thieme, 1933
Dietrich, A Herzmuskelschadigungen durch mittelbare Verletzungen im Kriege Arch f path. Anat, *237* 118, 1922
Dietrich, E Beitrag zur Diagnostik der Pulmonalsklerose Fortschr a d Geb d Rontgenstrahlen, *36* 990, 1927
Donzelot, E, Bardin, P, and Heim de Balsac, R Un nouveau cas de "diverticule du péricarde" Arch d mal d coeur, *37* 60, 1944
Donzelot, E, Eman-Zadé, A M, Heim de Balsac, R, Escale, J E, and Antoine, M L'angiocardiographie dans des cardiopathies congénitales Valeur Indications Semaine d hôp, *25* 2309, 1949
Doring, S Ueber linksseitige tonogene Herzdilatation im Tierexperiment. Ztschr f exper Med, *94* 766, 1934
Dormanns, E, and Emminger, E Untersuchungen uber die Beziehungen zwischen Weite der Aorta and Ausbreitung und Starke der Atherosklerose Arch f path Anat, *295* *225*, 1935
Dotter, C T, Hardisty, N M, and Steinberg, I Anomalous right pulmonary vein entering the inferior vena cava report of two cases diagnosed during life by angiocardiography and cardiac catheterization Am J M Sc, *218* 31, 1949
Dotter, C T, and Steinberg, I Angiocardiography Annals of Roentgenology, 1951, vol. 20
Dresler, K Beitrag zur Diagnose der Persistenz des Ductus arteriosus Botalli Mitt Verein. Schlesw Holst Aerzte, *11* 38, 1902
Dresler, K Nachtrag zur Diagnose der Persistenz des Ductus arteriosus Botalli Jahrb f Kinderheilk, *60* 54, 1904
Dressler, W Clinical Cardiology New York, Hoeber, 1942
Dressler, W Die Brustwandpulsationen als Symptome von Herz und Gefasskrankheiten, Wien· Maudrich, 1933
Dressler, W, and Fischer, R Ueber Trikuspidalestenose. Klin Wchnschr, *8* 1267, 1316, 1929.
Dressler, W, and Rosler, H Vorhofseptumdefekt kombiniert mit mitralstenose und aurikularem Leberpuls. Ztschr f klin Med, *112* 421, 1930
Duisenberg, C E, and Arismandi, L. The angiocardiographic demonstration of pulmonary arteriovenous fistula Radiology, *53* 66, 1949

Durieu, H, and Lequime, J Aspects radiologiques de la veine azygos au cours de l'insufficance cardiaque Arch d mal du. coeur, *31* 609, 1938

Duval, P, and Barnsby, B. Balle de fusil mobile dans le segment péricardiaque de la veine cave inférieure Extraction par péricardiotomie et incision de la veine cave Bull Soc Chir, *44* 1138, 1918.

E

Eakin, W. W, and Abbott, M. E Stenosis of the pulmonary conus at the lower bulbar orifice (conus, a separate chamber) and closed interventricular septum with two illustrative cases Am J M. Sc, *186* 860, 1933

Eck, Mannheimer, Ulsparre Morbus caeruleus Bibl cardiol Suppl ad Cardiologia, 1949, *4*.

Eckel, P Doppelaneurysmen der Aorta im Rontgenbilde Rontgenpraxis, *3* 784, 1931

Edeiken, J Effect of spinal deformities on the heart Am J M Sc., *186* 99, 1933

Edens, E Ueber Verschwartung des hintern Mittelfellraumes (Mediastinitis fibrosa post) Klin Wchnschr, *15* 332, 1936.

Edens, E Das kleine Herz Die Krankheiten des Herzens und der Gefasse, Berlin, 1939

Edwards, J E Retro-esophageal segment of the left aortic arch, right ligamentum arteriosum and right descending aorta causing a congenital vascular ring about the trachea and esophagus Proc Staff Meetings Mayo Clin, *23* 108, 1948

Egas Moniz, Lopo de Carvalho, and Almeida Lima La visibilite des vaisses aux pulmonaires aux rayons X par injection, dans l'oriclette droit, de fortes solutions d'iodine de sodium Bull Acad de méd, Paris, *105* 627, 1931

Eggli, A Eine Apparatur zur Auslosung herzphasensynchronisierter Thoraxaufnahmen zum Zwecke der Herzgrossenbestimmung und Lungenstereometrie Radiolog clin, *8* 129, 1939

Ehrenberg, L Zwei Falle von Tumor im Herzen, ein Beitrag zur Kenntnis der Pathologie und Symptomatologie der Herztumoren Dtsch Arch f klin Med, *103* 203, 1911

Eijkman, P Einschaltung der Rohre durch den Pulschreibhebel 81. Versammlung dtsch Naturforscher und Aerzte, Salzburg, 1909

Eimer, K Ergebnisse orthodiagraphischer Herzuntersuchungen bei Gepackmarschen Ztschr f exper Med, *60* 521, 1928

Eisenmenger, V Die angeborenen Defekte der Kammerscheidewand des Herzens Ztschr f klin Med, *32* 1, 1897

Eisler, F, and Kreuzfuchs, S Die Rontgendiagnose der Aortensyphilis Dtsch med. Wchnschr, *39* 2145, 1913

Elias, H, and Feller, A Stauungstypen bei Kreislaufstorungen mit besonderer Berucksichtigung der exsudativen Perikarditis. Wien, Springer, 1926

Elias, H, and Goldstein, J Zur Differenzierung des Stauungstypus bei Mitralstenose und Mitralinsuffizienz Wien Arch f inn Med, *18* 29, 1929

v Elischer, J. Ueber Momentrontgenbilder des gesunden und kranken Herzens in verschiedenen Phasen seiner Tatigkeit Ztschr f klin Med, *75* 45, 1912

Ellinger, E [illegible] 278, 1938

Emanuel, J [illegible]

v. Engelmaye [illegible]

Eppinger, E C, and Burwell, C S The mechanical effect of patent ductus arteriosus on the heart J A M A, *115* 1262, 1940

Eppinger, H. Zur Diagnostik eines wahren Aneurysmas des Sinus Valsalvae dexter. Wien med Wchnschr, *66* 65, 1916

Eppinger, H Das F [illegible]

Eppinger, H Kisch [illegible] grosse durch [illegible] Wchnschr, *5* 781, 1926

Eppinger, H., Kisch, F, and Schwarz, H: Das Versagen des Kreislaufs. Berlin, Springer, 1927.

[illegible] Entzundung Wien, Springer, *1935*.

[illegible] der Aorta in der klinischen *Diagnostik*. [illegible] 927.

Erdélyi, J. Neues Verfahren zum Nachweis der Wandverhartung des Aortenbogens Klin Wchnschr, *8* 260, 1929
Erdheim, J. Neuere Fortschritte in der Gefasspathologie Wien klin Wchnschr *49* 1399, 1936
Ernst, F. Ein Beitrag zum entzundlichen Herzbeuteldivertikel Rontgenpraxis, 7 324, 1935
Ernstene, A C., and Robins, S A Roentgenological diagnosis of stenosis of descending arch (coarctation of the aorta). Am J Roentgenol, *25* 137, 1931
Eskuchen, E. Beitrage zur Klinik des Bauchaortenaneurysmas mit Angabe besonderer radioskopischer Merkmale Klin Wchnschr, *2* 2202, 1923
Esser, A Seltene Formen von Aneurysmen Ztschr f Kreislaufforsch, *24* 227, 1932
Ettinger, A, Magendantz, H, and Russo, E A Arteriovenous aneurysm of the lung Radiology, *53* 261, 1949
Evans, C Myxoedema heart disease Treatment and radiologic changes Lancet, *2* 1300, 1937
Evans, W The heart in sternal depression Brit Heart J, *8* 132, 1946
Exalto, J, Dicke, W. K, and Aalsmeer, W C Congenital stricture of trachea and esophagus by double aortic arch Arch chir Neederland, *2* 170, 1950
Eyster, J A E Determination of cardiac hypertrophy by roentgen-ray methods Arch Int Med, *41* 667, 1928

F

Fahr, G Myxedema heart disease J.A M A, *84* 345, 1925
Fahr, Th Verhalten des Herzens und die Herzkrankheiten Handb arztl Erfg, Weltkrg 1914-18 Bd VIII, 1921
v Falkenhausen, M Zur Rontgendiagnose der Aortitis luica im unteren Teil der Aorta thoracica Fortschr a d Geb d Rontgenstrahlen, *38* 672, 1928
Fanconi, G Zur Diagnose der Pleuritis mediastinalis fibrosa im Kindesalter Die "pleuritischen" Mediastinalstreifen Rontgenpraxis, *3* 49, 1931
Fanconi, G · Die Transposition der grossen Gefasse (Das charakteristische Rontgenbild). Arch Kinderheilk, *95* 20, 1932
Farmer, H. L Abdominal aneurysm with report of three cases Am J Roentgenol *18* 550, 1927
Favorite, G O Cor biatriatum triloculare with rudimentary right ventricle, hypoplasia of transposed aorta and patent ductus arteriosus, terminating by rupture of the dilated pulmonary artery Am J M Sc, *187* 663, 1934
Feer, E. Kropfherz und Thymusherz der Neugeborenen und Sauglinge Mschr Kinderheilk, *25* 88, 1923
Feldman, M Calcification of the abdominal aorta Radiology, *23* 700, 1934
Fetzer, H Ein atypischer Fall von Aortenaneurysma Fortschr a d Geb d Rontgenstrahlen, *37* 70, 1928
Fetzer, H Die Rontgendiagnostik der Aortenerkrankungen Rontgenpraxis, *1* 523, 1929
Fetzer, H Die Anwendung der Rontgenkymographie in der Kreislaufdiagnostik Ergebn d inn Med, *45* 485, 1933
Fick, W Kreislaufwirkung arteriovenoser Aneurysmen Dtsch Ztschr f Chir, *240* 113, 1933
Finkh, F. Die Rontgendiagnose von Steckschussen des Herzens Beitr z klin Chir, *98* 484, 1916
Fingerhuth, M, and Bickenbach, O Beitrag zur Klinik der Trikuspidalinsuffizienz Dtsch Arch f klin Med, *175* 577, 1933
Fischer, H Zur Frage der schwieligen Perikarditis, ihrer Erkennung und Behandlung Schweiz med Wchnschr, *56* 489, 1926
Fischer, R Fall von isolierter, relativer Trikuspidalinsuffizienz Wien klin Wchnschr, *41* 647, 1928, *46* 1546, 1933
Fischer, U Der Kreislauf unter Beschleunigung Rontgenaufnahmen beim Affen Luftl med, 1937, 2, cited after Zentralbl f Radiol, *27* 1938
Fleischner, F Fall von Verkalkung des Annulus fibrosus Wien med. Wchnschr, *75* 2721, 1925
Fleischner, F Korrigierte Kreuzfuchssche Messung Fortschr. a d Geb d. Rontgenstrahlen, *34* 426, 1926
Fleischner, F Lagebeziehungen von Oesophagus und Aorta bei hochgradiger Erweiterung der Aorta Fortschr a d Geb d Rontgenstrahlen, *41* 289, 1930

Forgacs, P. Congenital heart diseases with isolated inversion of abdominal viscera Brit. Heart J, 9 27, 1947

Forssmann, W. Die Sondierung des rechten Herzens. Klin Wchnschr, 8 2085, 1929

Forssmann, W. Ueber Kontrastdarstellung der Hohlen des lebenden rechten Herzens und der Lungenschlagader Munchen. med Wchnschr, 78 489, 1931

Franchini, F Osservazioni intorno al movimento sistolico diastolico ed allo spostamento inspiratorio di proiettili infitti en cuore Radiol med, 7 116, 1920

Frank, M Zur Frage der Varixbildung im rechten Vorhof Zentralbl f allg Path, 31 205, 1920/21

Franke, H. Klinischer Beitrag zur Stenose des Arcus aortae Dtsch Arch f klin Med, 186 304, 1940

Franke, M Beitrage zur Nephritisfrage. I Ueber das Verhalten des Herzens und der grossen Gefasse bei der akuten Nephritis Dtsch. Arch f klin Med, 22 428, 1927.

Fray, W W., The roentgenological diagnosis of coarctation of the aorta (adult type) Am J Roentgenol, 24:349, 1930

Fray, W W Mensuration of the heart and chest in the left posteroanterior oblique position A comparative study I Relation of the transverse diameter of the heart to the thorax II Determination of type of cardiac enlargement (right or left) Am J Roentgenol, 27 177, 363, 1932

Fray, W W Right aortic arch Radiology, 26 27, 1936

Fredzel, G, Lind, J, Ohlson, E, and Wegelius, C Direct serial roentgenography in two planes simultaneously at 0 08 second interval Am J Roentgenol, 63 548, 1950

Freedman, E The roentgenological diagnosis of cardiac compression due to pericardial scar Am J Roentgenol, 37 739, 1937

Freedman, E Inflammatory diverticula of the pericardium Am J Roentgenol, 37 733, 1937

Freeman, E B Chronic pericardial effusion in myxedema Ann Int Med, 7 1070, 1934

Freund, R, and Caspersohn, C Schrapnellkugel in der rechten Herzkammer, Munchen med. Wchnschr, 62 1199, 1915

Frey, W Das Verhalten des Herzgefasssystems bei der Kompression arteriovenoser Aneurysmen Munchen med Wchnschr, 66 1106, 1919

Friedlander, C Ueber Panzerherz Fortschr a d Geb d Rontgenstrahlen, 34 145, 1926

Frik, K Zur Deutung des Rontgenbildes im ersten schragen Durchmesser Fortschr a d Geb d Rontgenstrahlen, 29 728, 1922

Frik, K Die normale Aorta im Rontgenbild. Fortschr a d Geb d Rontgenstrahlen, 33 29, 1925

Frik, K Form- und Funktionsanderungen des Herzens im Röntgenbild. X. Fortbild-Lehrg in Bad Nauheim, 1934

Fuchs, G Das Rontgenbild des Aneurysmas der Arteria lienalis. Rontgenpraxis, 9 467, 1937

Fuchs, G Seltenere Veranderungen der grossen Gefasse im Rontgenbild. Tib. fakultesi, 1940

G

Gabert, E Die hintere Herzrand im Röntgenbild in normalen und kranken Fallen und Veranderungen des Tracheobronchialbaumes durch Erweiterung des linken Vorhofes Fortschr a d Geb d Rontgenstrahlen, 32 385, 1924

Gabert, E Die Lagebeziehungen des Oesophagus zur hinteren Herzflache und ihre Veranderungen durch Erweiterung des linken Vorhofes im Rontgenbild Fortschr a d Geb d Rontgenstrahlen, 32 410, 1924

Gaisbock, F Bruststeckschüsse mit Schädigung des Herzens und deren Verlauf Wien klin Wchnschr., 30 1610, 1917.

Gander, G Ein Beitrag zur Frage der verastelten Lungenverknocherungen Fortschr a d Geb. d Röntgenstrahlen, 44 448, 1931

Gasul, M. B, Fell, E. H., and Casas, R.: The diagnosis of aortic septal defect by retrograde aortography Circulation, 4 251, 1951.

Gassul, R.: Ueber einen offenen Ductus Botalli mit Beteilugung des linken Herzens. Dtsch med Wchnschr., 47:559, 1921.

Gatersleben, H Ein Beitrag zu den Milzcysten Dtsch Ztschr f Chir., 212 139, 1928

Gefferth, K · Beitrage zur Röntgendiagnostik der kongenitalen Herzfehler auf Grund von sechs durch Sektion kontrollierten Fallen Monatschr. f Kinderh 85-250, 1941
Geigel, R. Die klinische Verwertung der Herzsilhouette Munchen med. Wchnschr. 61 1220, 1914
Geigel, R. Herzgrosse und Wehrkraft Munchen med Wchnschr, 63 953, 1916
Geigel, R · Das pulsierende Herz. Munchen med Wchnschr, 67 1316, 1920
Geigel, R. Der reduzierte Herzquotient Munchen med Wchnschr., 67 728, 1920
Geigel, R Die klinische Bedeutung der Herzgrosse und des Blutdrucks Ergbn inn Med, 20 192, 1921
Geipel, P. Weitere Beitrage zum Situs transversus und zur Lehre von den Transpositionen der grossen Gefasse des Herzens Arch f Kinderheilk, 35 118, 122, 1903
Geipel, P. Zur Verkalkung der Herzmuskelfaser Fortschr a.d Geb d Röntgenstrahlen, 34 311, 1926
Gerhartz, H Die diagnostische Bedeutung der rontgenologisch sichtbaren Lungengefasspulsationen Zentralbl f Herzkrankh, 7 262, 1915
Gerhartz, H Das Rontgenbild des Aortenstenosenherzens. Med Klin, 20 742, 1924.
Gerlach, W Postmortale Form- und Lageanderungen mit besonderer Berücksichtigung der Totenstarre Ergebn d allg Path u path Anat, 20 (2), 259, 1923
Gerstmann, H Ein Fall von Aortenventrikel Arch f path Anat, 271 1, 1929.
v Giercke, E Hepato-Nephromegalia glykogenica (Glykogenspeicherkrankheit der Leber und Niere) Beitr z path Anat a z allg Path, 82 497, 1929
Giordano, G Ulteriore contributo allo studio della sinistrocardia posteriore acquista-particolare deformazione della trachea Radiol med, 25 820, 1938
Gluck, A Seltener Sitz eines Aortenaneurysmas Sitz d Wien, Rontgenges, 1948
Goedel, A Eine ungewohnliche Form der Herzvergrosserung (enorme Vorhofvergrösserung) bei Mitralstenose Wien klin Wchnschr, 42 427, 1929
Golden, A, and Weems, H S The diagnosis of dissecting aneurysm of the aorta by angiocardiography Am Heart J, 37 115, 1949
Goldenberg, M, and Zdansky, E Ueber die akute Herzverweiterung im Coronarkrampf Klin. Wchnschr, 11 1498, 1932
Goldscheider Ueber die Untersuchung des Herzens in linker Seitenlage Dtsch med. Wchnschr, 32 1649, 1906
Golonsko, R Ein kasuistischer Beitrag zur Erkennbarkeit des Herzaneurysmas im Roentgenbild. Rontgenpraxis, 1 694, 1929
Gordon, A H Some clinical aspects of hypothyroidism Candad M A. J, 20 7, 1929
Gordon, B, and Strong, G F Studies on the rabbit's heart II Effect of vigorous exercise on the size of the normal and abnormal heart Arch Int Med, 32 517, 1923
Gott, Th., and Rosenthal, J Ueber ein Verfahren zur Darstellung der Herzbewegung mittels Röntgenstrahlen (Rontgenkymographische Untersuchungen des Herzens) Fortschr a d. Geb. d Rontgenstrahlen, 59 2033, 1929
Graham, E A Aneurysm of the ductus arteriosus with considerations of its importance to the thoracic surgeon Arch Surg, 41 324, 1940
Grashey, R Verkalkte Mesenterialgefasse Acta radiol, 6 466, 1926
Gravinghoff, W Anfallsweise Cyanose bei angeborenem Herzfehler Monatschr f Kinderh, 35 242, 1927
Grävinghoff, W Die Rontgenanatomie der Brust- und Bauchorgane, in Handbuch der Anatomie des Kindes, Bd I, Munchen, Bergmann, 1934
Greineder, K Die umklammernde hohe Rechtlage des Aortenbogens und ihre differentialdiagnostische Bedeutung Fortschr a d Geb d Rontgenstrahlen, 57.535, 1938
Grisebach, M Wie weit lasst sich die Herzcontour im Röntgenschirmbild mit den absoluten Herzmassen im Orthodiagramm in Einklang bringen? Fortschr. a d Geb d Rontgenstrahlen, 66 24, 1943
Grishman, A, Steinberg, M F, and Oppenheimer, B. S. The clinical diagnosis of idiopathic dilatation of the pulmonary artery J Mt. Sinai Hosp, 10 142, 1943.
Grishman, A, Steinberg, M F, and Sussman, M L. Contrast visualization of the coarctation of the aorta Am Heart J, 21 365, 1941.

Grishman, A, Steinberg, M F, and Sussman, M. L Tetralogy of Fallot, contrast visualization of heart and great vessels Radiology, *37* 178, 1941
Grishman, A, Steinberg, M F, and Sussman, M L Congenital aortic and subaortic stenosis with associated anomalies of the aorta Med Clin North America, *31* 543, 1947.
Grishman, A, Sussman, M L, and Steinberg, M F Atypical coarctation of the aorta with absence of the radial pulse. Am Heart J, *27* 217, 1944
Grishman, A, Sussman, M L., and Steinberg, M F Angiocardiographic analysis of the cardiac configuration in rheumatic mitral disease Am J Roentgenol, *51* 33, 1944
Grob, M Ueber Anomalien des Aortenbogens und ihre entwicklungsgeschichtliche Genese Helvetica Paed Acta, *4* 274, 1949
Grob, M, Rossi, E, and Bettex, M, Zur Diagnose der angeborenen Pulmonalstenosen mit Vorhofseptumdefekt Helvetica Paed Acta, *5* 345, 1950
Grober, J Untersuchungen zur Arbeitshypertrophie des Herzens Dtsch Arch f klin Med, *91* 502, 1907
Groedel, F M Erste Mitteilung uber die Differenzierung einzeler Herzhohlen im Rontgenbilde und den Nachweis von Kalkschatten in der Herzsilhouette intra vitam Fortschr a d Geb d Rontgenstrahlen, *16* 337, 1910/11
Groedel, F M Das Verhalten des Herzens bei kongenitaler Trichterbrust Munchen med Wchnschr, *58* 684, 1911
Groedel, F M Die rontgenanatomische Situsuntersuchung des Herzens und der grossen Gefasse Zweck, Bedeutung und seitherige Leistungen dieses Verfahrens Dtsch Arch f klin Med, *111* 192, 1913
Groedel, F M Zur Rontgenuntersuchung des Herzens bei fraglicher Militardiensttauglichkeit Munchen med Wchnschr, *62* 1781, 1915
Groedel, F M Die Dimensionen des normalen Aortenorthodiagrammes Berl klin Wchnschr, *55* 327, 1918
Groedel, F M Vereinfachte Ausmessung des Herzorthodiagrammes nach Theo. Groedel Munchen med Wchnschr, *65* 397, 1918
Groedel, F M Ist das Herz im perikardischen Exsudat rontgenologisch darstellbar? Fortschr a d Geb d Rontgenstrahlen, *27* 32, 1919/21.
Groedel, F M Die Rontgenologische Untersuchung des kindlichen Herzens Ztschr f Kinderh, *29* 36, 1921
Groedel, F M Wie verhalt sich das vergrosserte Herz im wachsenden Korper? Arch f Kinderh, *69* 365, 1921
Groedel, F M Was leistet das Rontgenverfahren fur die Funktionsprufung des Herzens? Dtsch Arch f. klin Med, *138* 145, 1922
Groedel, F M Der rontgenologische Nachweis des Herzneurysmas Munchen med Wchnschr, *80* 210, 1933
Groedel, F M Die Rontgenuntersuchung des Herzens. In Lehrbuch und Atlas der Rontgendiagnostik in der inneren Medizin und ihren Grenzgebieten Munchen, Lehmann, 1938
Groedel, F M Aneurysm of the pulmonary artery Radiol, *33* 219, 1939
Groedel, Th, and Groedel, Fr Ueber die Form der Herzsilhouette bei den angeborenen Herzkrankheiten Dtsch Arch f klin Med, *103* 413, 1911
Groedel, Th, and Groedel, Fr Kombinierte rontgenkinematographische und elektrokardiographische Herzuntersuchung Dtsch Arch f klin Med, *109* 52, 1912.
Grollman, A, and Baumann, A Schlagvolumen und Zeitvolumen bei gesunden und kranken Menschen. Dresden, Steinkopff, 1935
Gross, A Knotige Knochenbildung bei chronischer kardialer Lungenstauung Fortschr a d Geb. d Rontgenstrahlen, *58* 33, 1938
Gross, L Lesions of the left auricle in rheumatic fever Am J Path, *11*:711, 1935.
Gross, R. E Surgical Treatment for Abnormalities of the Heart and Great Vessels Springfield, Thomas, 1947.

correction and report New England Med J, *233* 287, 1945.

Grothusen. Ueber Verkleinerung des Herzens nach Aderlass Med Klin., *25* 265, 1929
Grunberg, F. W. Ueber einige Veranderungen von seiten des Herzgefasssystems bei schweren Anamien. Dtsch Arch f inn Med, *169* 354, 1930
Guggenheim, A Aneurysma des Ductus Arteriosus Botalli mit Ruptur Frankf Ztschr f Path, *40* 436, 1930

H

Haas, L. Diverticulum pericardii Acta radiol, *20* 228, 1939
Hackensellner, H A. Beitrag zur Kasuistik und Genese der sog Trikuspidalatresie Klin Med, *6* 218, 1951
Hadorn, W, and Tillmann, A. Ueber Contusio cordis Ztschr f Kreislaufforsch, *28* 185, 1936
Haenisch, F. Zur Röntgendiagnose des Aneurysma der Aorta descendens Fortschr a d Geb d Röntgenstrahlen, *30* 523, 1923
Hall, A S Relation of the pulmonary artery and the esophagus Lancet, *2* 18, 1933
Haffner, J Fall von verkalktem Aneurysma der Arteria lienalis Acta radiol, *17* 602, 1936
Hammer, G Die rontgenologischen Methoden der Herzgrossenbestimmung Fortschr a d Geb d Rontgenstrahlen, *25* 510, 1918
Hammer, G Situs inversus arcus aortae Fortschr a d Geb d Rontgenstrahlen, *34* 517, 1926
Hammer, G Die Herzflache als Massstab fur die Herzgrossenbestimmung Fortschr a d Geb d Rontgenstrahlen, *38* 1000, 1928
Hammer, G, and Mies, H Rontgenographische Untersuchungen, inbesondere des Herzens nach Dauerausschaltung der Blutdruckzugler Krankh forschg, *7* 257, 1929
Hampton, A O, and Castleman, B Correlation of postmortem teleroentgenograms with autopsy findings, with special reference to pulmonary embolism and infarction Am J Roentgenol, *43* 305, 1940
Hanke, R Ueber einen Fall von rechtslaufiger Aorta mit tiefem Ursprung der Art subclavia sin Ztschr f Kreislaufforsch, *33* 241, 1941
Hare, D C, and Ross, J M Syphilitic disease of the pulmonary arteries Lancet, *2* 806, 1929
Hart, K Frage der Plethora vera Dtsch med Wchnschr, *38* 798, 1912
Hartung, A Zur Diagnose des Bauchaortenaneurysmas Rontgenpraxis, *7* 395, 1935
Hasebrock Ueber die Arbeitshypertrophie des Herzens Dtsch Arch f klin Med, *131* 62, 1920
Hashimoto, H Acute pernicious form of beriberi and its treatment by intravenous administration of Vitamin B_1 Am Heart J, *13* 580, 1937
Hasselwander, A Beitrage zur Methodik der Rontgenographie I Die Telerontgenographie. Fortschr a d Geb d Rontgenstrahlen, *19* 356, 1912/13
Haubrich, R Ueber die einseitige Lungenstauung Fortschr a d Geb d Rontgenstrahlen, *71* 571, 1949
Haudek, M Eine Revision der Methodik der rontgenologischen Herzgrossenbeurteilung Jahrk. arztl Fortbildung, *9* 8, 1918
Haug, K, and Jaenisch, R Ueber experimentell erzeugte akute Veranderungen der Herzgrosse beim Menschen Ztschr f klin Med, *114* 733, 1930
Hecht, H F Die Verwertung des orthodiagraphischen Herzflachenmessung fur die Beurteilung der Herzgrosse im Kindesalter Jahrb f Kinderh, *133* 26, 1931
v Hecker, H Ausgedehntes "Panzerherz" als Zufallsbefund Fortschr a d Geb d Rontgenstrahlen, *31* 264, 1923/24
Heckler, H Ein seltener Fall von zwei intraperikardial gelegenen Aortenaneurysmen (Aneurysma der Sinus Valsalva) Rontgenpraxis, *8* 81, 1936
Heckmann, K Ueber Herzkymographie Munchen med Wchnschr, *82* 1079, 1935
Heckmann, K Kymographische Untersuchungen normaler und pathologischer Aktionsformen der Ventrikel Klin Wchnschr, *14* 700, 1935
Heckmann, K Ein Verfahren zur Untersuchung der Pulsation des Herzens und anderer Organe mittels Rontgenstrahlen Klin Wchnschr, *13* 13, 1936
Heckmann, K Moderne Methoden zur Untersuchung der Herzpulsationen mittels Rontgenstrahlen Ergebn inn Med u Kinderheilk, *52* 536, 1937.
Heckmann, K Die Untersuchung der Herzpulsationen mittels Elektrokymographie und Phasenanalyse Ztschr f Kreislaufforsch, *41* 144, 1952.

Heckmann, K Pathologische Pulsationformen der Ventrikel Fortschr a d Geb d Röntgenstrahlen, 76 175, 1952
Heckmann, K Ueber ein Perikardphanomen bei linkseitigem Pneumothorax und durch Flussigkeitsbewegung im Herzbeutel verursachte kymographische Befunde Klin Wchnschr, 14 1422, 1935
Heckmann, K Zur Frage der Doppelgipfeligkeit der Randzacken in Flachenkymogramm Klin Wchnschr, 15 644, 1936
Heckmann, K Ein Verfahren zur Untersuchung der Pulsationen des Herzens und anderer Organe mittels Rontgenstrahlen Ein versuch zur Registrierung von Volumenanderungen der Organe wahrend der Herzpulsationen (Vorlaufige Mitteilung) Klin Wchnschr, 15 13, 1936
Heckmann, K Die Lageanderungen des Herzens wahrend der Pulsation und ihr Ausdruck im Flachenkymogramm Fortschr a d Geb d Rontgenstrahlen, 55 319, 1937
Heckmann, K. Die Symptome des Perikardergusses Munchen med Wchnschr, 84 60, 1937
Ges Path, 11 303, 1907
iovenose Lungenaneurysmen und ihre
Wchnschr, 16 367, 1951
Hedvall, E Arterio-venous aneurysm in the lung Acta tuberculosa Suppl XXI, 32, 1949
Heeren, J Zur Rontgendiagnose verkalkter Herzthromben Fortschr a d. Geb d Rontgenstrahlen, 50 490, 1935
Heim de Balsac, R Das rechte Vorhof Eine anatomisch-rontgenologische Studie an Leichen mittels Kontrastmittel Fortschr a d Geb d Rontgenstrahlen, 57 73, 1938
Heim de Balsac, R *Principle, technique et pratique de la radiokymographie cardiovasculaire* Semaine de hôp, 24 2278, 1948
Heim de Balsac, R, and Marquis, G Un nouveau d'anévrisme de coeur calcifié Arch mal du coeur, 34 142, 1941
Heimberger, H. Ueber Panzerherz Fortschr a d Geb d Rontgenstrahlen, 32 46, 1924
Heitz and Corone Du diagnostic radiologique de l'anévrisme parietal du coeur Arch d mal du coeur 16 494, 1923.
Hellmer, H Fall von "primarer Dextroversion" des Herzens (sog korrigierte Transposition nach Rokitansky) Fortschr. a d Geb d Rontgenstrahlen, 51 591, 1935
Henderson, Y Atelektase und Massiver Lungenkollaps und verwandte postoperative Zustande Munchen med Wchnschr, 83 305, 1936
Heninger, B R Clinical aspects of pericardial metastasis Ann Int Med, 7 1359, 1934
Henny, G C, and Boone, B R Electrokymograph for recording heart motions utilizing the roentgenoscope Am J Roentgenol, 45 217, 1945
Henschen, S E *Das Aneurysma arteriae pulmonalis Sammlg klin Vortr* N F Nr 422/423, 1906
Herbst, R Herzgrosse und Luftdruckverminderung Verhandl d dtsch Ges f Kreislaufforsch, 1936, 290
Herrman, W G Pulmonary changes in a case of periarteritis nodosa Am J Roentgenol, 29 607, 1933
Herrnheiser, G Die Tiefenlage der im Orthodiagramm randbildenden Herz-Gefasspartien Fortschr a d Geb d Rontgenstrahlen, 28 372, 1921/22
Herrnheiser, G, and Kubat, A *Systematische Anatomie der Lungengefasse* Ztschr Anat u
Hert 'asten, mit
Herx Ztschr f. klin. Med, 96·218, 1923.
d Geb
ortschr.
a d Geb d Rontgenstrahlen
Hess, L Ueber konstitutionelle Herzveranderungen. Ztschr f Konstit. Lehre, 9 72, 1923.
Hess, W R · Die Regulierung des Blutkreislaufes, Leipzig, 1930

Hesse, E Aneurysma aortae abdominalis Fortschr a d Geb d Rontgenstrahlen, *14*,130, 1909/10
Hessmann, A, and Israelski, M, "Panzerherz" Rontgenpraxis, *4* 112, 1932.
Hippe, H, and Hahle, K Zum Lobus venae azygos Rontgenpraxis, *8* 95, 1936
Hirsch, J S Examination of the heart by roentgenkymographic method. Brit J. Radiol (N ser) *7* 728, 1934
Hirsch, J S, and Schwarzschild, M Directed roentgenography of the thorax (The cardiocariograph) Am J Roentgenol, *37* 13, 1937
Hirsch, K Ueber die Beziehungen zwischen dem Herzmuskel und der Korpermuskalatur. Dtsch. Arch f. klin Med, *64*:597, 1899, *68* 55, 321, 1900
Hitzenberger, K Die pulsatorischer Bewegungen des rechten Zwerchfells Wien Arch f inn Med, *9* 205, 1925
Hitzenberger, K., Das Zwerchell in gesunden und kranken Zustand Wein, Springer, 1927
Hitzenberger, K Die Klappenfehler im Rontgenbild, Klin Wchnschr, *7* 850, 1928
Hitzenberger, K, and Elias, H Beitrage zur vergleichenden Krankenuntersuchung durch physikalisch-klinische und rontgenologische Methoden Wien Arch f inn Med, *7* 355, 1924
Hitzenberger, K., and Reich, L Ein Beitrag zur Rontgenkymographie Fortschr a d Geb d Rontgenstrahlen, *31* 17, 1923
Hjelm, R, and Hultén, O Rontgenologische Studien uber den Lobus der Vena azygos Acta radiol, *9* 126, 1928.
Hochrein, M Neuere Beobachtungen am venosen und pulmonalen Kreislauf Munchen med Wchnschr, *80* 1729, 1933
Hochrein, M., and Keller, C J Beitrage zur Blutzirkulation im kleinen Kreislauf I and II Mitteilung Arch f exper Path u Pharmakol, *164* 529, 552, 1932
Hochrein, M, and Matthes, K Die Kreislauffunktion beim Sport Arzt, und Sport I (1935) (In Dtsch med Wchnschr, 1935)
Hochrein, M, and Matthes, K Ueber die Beziehungen des Drucks im linken Vorhof zur Blutgehalt der Lunge. Arch f d ges Physiol, *233* 1, 1923
Hochstadt, O, and Silbermann, J Ueber einen besonderen Fall von persistierendem Ductus arteriosus Botalli Radiol clin, *9* 192, 1940
Hodges, P C Comparison of the teleroentgenogram with the orthodiagram Am J Roentgenol., *11* 466, 1924
Hodges, P C, and Eyster, J A E Estimation of transverse cardiac diameter in man Arch Int, Med, *37* 707, 1926, Estimation of the cardiac area in man Am J Roentgenol, *12* 261, 1924
v Hoesslin, H Das kleine Herz der Asthmatiker Klin, Wchnschr, *10* 1893, 1941
Hofbauer, L Extrakardiale Kreislaufstorungen Ergebn inn Med, *49* 464, 1935
Hofbauer, L Kreislaufschwache bei Kyphoskoliose Demonstration Ges d Aerzte, *Wien*, *11* 1, 1935
Hofbauer, L, and Holzknecht, G Respiratorische Grossenschwankungen des *Herzen Mitt* Labor, radiol Diagn u Ther a g Krk hs Wien, 1907.
Hoffmann, A, Herz und Konstitution Jahrk aerztl Fortbildg, *9* 3,1918
Hollmann, W Aneurysma der Bauchaorta Rontgenpraxis, *6* 811, 1934
Holmes, G The radiographic findings in pericarditis with effusion Am J *Roentgenol*, *7* 7, *1920*.
Holmes, G Some observations on use of roentgen rays in diagnosis of *pericarditis* J A M A, *83* 1745, 1924
Holmes, G Congenital idiopathic enlargement of the heart Am J *Roentgenol 25* 120, *1931*
Holst, L Die Erweiterung des Pulmonalisbogens im Rontgenbilde (Funf Falle von Aneurysma der Pulmonalarterie) Fortschr a d Geb d Rontgenstrahlen, *50* 122, 1934
Holzapfel, G Ungewohnlicher Verlauf der Arteria subclavia dextra, Anat H, I Abt Arb anat Inst, *12* 309, 1899
Holzknecht, G Das radiographische Verhalten der normalen *Aorten Wien* klin Wchnschr, *13* 225, 1900,
Holzknecht, G Zum radiologische Verhalten pathologischer Prozesse *der Brustaorta* Wien klin Wchnschr, *13* 573, 1900
Holzknecht, G Die rontgenologische Diagnostik der Erkrankungen *der Brusteingeweide*. Hamburg, Grafe und Sillem, 1901

Holzmann, M. Aneurysma dissecans der Bauchaorta im Rontgenbild. Acta radiol, *13*·21, 1932

Holzmann, M. Rontgenbefunde bei Trikuspidalfehlern Fortschr a d. Geb d Rontgenstrahlen, *46* 14, 1932.

Holzmann, M Erkrankungen des Herzens und der Gefasse. Lehrbuch der *Rontgendiagnostik von* Schinz, Baensch, u Friedel, Leipzig, Thieme, 1939

Holzmann, M. Erkrankungen des Herzens und der Gefasse. Lehrbuch der Rontgendiagnostik von Schinz, Stuttgart, *1952.*

Hormuth, V. Aortenruptur mit Brustwandhamatom bei sackartigem Aneurysma der Aorta ascendens Rontgenpraxis, *9* 609, 1937

Horneck, K. Zur Kenntnis der ubermassigen Erweiterung des linken Vorhofes Wien klin Wchnschr., *47* 1348, 1934

Horsch, K. Zur Frage der Pericarditis calculosa Mitt a d. Grenzgeb Med u Chir., *43* 370, 1933.

Hoskin, J, and Gardner, F Silent dissection of the aorta. Brit *Heart J*, *8 141, 1946*

Hotz, A. Ueber angeborene Trikuspidalinsuffizienz Jahrb. Kinderh, *102* 1, 1923

Hotz, A. Besonderheiten der kindlichen Brustorgane. In Schinz, Baensch, and Friedl Lehrbuch der Rontgendiagnostik, Leipzig, Thieme, 1939

Huber, H., Ueber erworbene Defekte der Herzkammerscheidewand Ztschr f Kreislaufforsch, *27* 825, 1935.

Hubert, G Die Aortitis syphilitica im Rontgenbild Fortschr a d Geb. d Rontgenstrahlen, *33* 36, 1925

Huismans, L Der Telekardiograph, ein Ersatz des Orthodiagraphen Munchen med Wchnschr, *60* 2400, 1913.

Huismans, L Eine einfache Methode, die "Herzspitze" fur die Messung des Langsdurchmessers des Herzens sichtbar zu machen Dtsch med Wchnschr., *40* 1429, 1914

Huismans, L Die Telekardiographie Ztschr f klin. Med, *85*·33, 1918.

Humphreys, E M Truncus arteriosus communis persistens Criteria for identification of common arterial trunk, with report of case with four semilunar cusps. Arch Path, *14* 1932, 671

Hurxthal, L M Myxedema heart with congestive heart failure and polyserous effusion New England J. Med., *213* 264, 1935

I

Ickert, F Der Blutdruck bei Ermudung und Infekten Dtsch med Wchnschr, *69* 7, 1943.

Ickert, F Die Erschopfung Med Klin, *38* 280, 1943

Ihre, B Roentgenography ad modum Stumpf as a method examining the heart Acta radiol, *15* 107, 1934.

Israelski, M Die verkalkte Arteria lienalis im Rontgenbilder Rontgenpraxis, *2* 670, 1930

Israelski, M., and Lukas, E. Klinische und rontgenologische Beobachtungen an Lungen und Herz nach Leuchtgasvergiftung Klin Wchnschr, *9* 978, 1930

Israelski, M, and Lukas, E Ueber akute Herzerweiterung, Herzbeutelerguss und ihre Differentialdiagnose Klin Wchnschr, *10* 2301, 1931

J

Jacchia, P. Phlebektasie im Lungenparenchym Acta radiol, *17* 74, 1936

Jackson, F The radiology of acute pulmonary edema Brit Heart J., *13* 503, 1951.

Jacobson, V C Deviation of the aortic septum complete transposition of the great vessels Am J Dis Child, *21* 176, 1921

Jaderholm, K. B Ueber Perikarddivertikel Acta chir Scandinav., *71* 517, 1932

Jaffé, R. H, and Sternberg, H Ueber die physiologischen Schwankungen des Aortenumfanges. Med Klin, *15* 1311, 1919

v. Jagič, M, and Kreuzfuchs, S Zur Perkussion der Aorta auf der vorderen Brustwand unter Rontgenkontrolle Med Klin, *17*,1262, 1921

Jahn, J Stoffwechsel und Kreislauf bei der Dekompensation des Herzens. Klin Wchnschr, *14*·231, 1935.

Jaksch-Wartenhorst, R. Herzaneurysma im Rontgenbild Fortschr a d Geb d. Rontgenstrahlen, *33*·563, 1935.

Jalet, J Des diverses erreurs, que l'on peut commettre au cours de la recherche de l'indice des Vaquez-Bordet, et de leur importance relative Arch d Electr méd, *37* 267, 1929

Jamin, F, and Merkel, H Die Koronararterien des menschlichen Herzens unter normalen und pathologischen Verhaltnissen Jena, G Fischer, 1907

Janker, R Knotige Knochenbildungen der Lungen Fortschr a d Geb d Rontgenstrahlen, *53* 260, 1936

Janker, R Ein rontgenkinematographischer Film uber die Kontrastdarstellung der Herzbinnenraume und der grossen Gefasse bei angeborenen Herzfehlern Langenbecks Arch u Dtsch Ztschr. f Chir, *266* 322, 1950

Janker, R, and Hallerbach, H Die rontgenkinematographische Darstellung der Trikuspidalatresie Fortschr. a d Geb d Rontgenstrahlen, *75* 31, 1951

Jansson, G Beitrag zur Rontgendiagnostik beim Perikarddivertikel Acta Radiol, *12* 50, 1931

Jarisch, A, and Loos, H O Ueber die Formveranderungen des Herzens bei experimentellen Herzerweiterungen Wien klin Wchnschr, *44* 844, 1931

Jaubert de Beaujeau, A, and Bège, R Situs inversus total avec lésions congénitales du coeur Arch d mal du coeur, *30* 977, 1937

Jellen, J, and Fischer, W F Intrapericardial teratoma Am J Dis Child, *51* 1397, 1936

Jenckel Schuss in den Herzbeutel Med Klin, *11* 68, 1915

Jensen, F G, and Norgaard, N Récherches cliniques, roentgénologiques et électrocardiographiques sur les femmes saines au cours de la grossesse normale Compt rend Soc Biol, *94* 1213, 1926

Jensen, G Beitrag zu dem klinisch-radiologischen Bild der Transposition grosser Gefasse und zur Theorie Spitzers von ihrer Entstehung Frankf Ztschr f Path, *43* 546, 1932

Jones, A M, and Langley, F A Chronic dissecting aneurysms Brit Heart J, *8* 191, 1946

Jones, E W A radiographic study of the coronary arteries in health and disease Quart J Med, *24* 199, 1931

Jones, J C, and Thompson, W P Arteriovenous fistula of the lung J Thoracic Surg, *13* 357, 1944

Jonsson, C, Broden, B, and Karnell, J Thoracic aortography Acta radiol, Suppl *89* 1, 1951

Jonsson, G, and Saltzman, G F Infundibulum of the patent ductus arteriosus studied by thoracic aortography Acta radiol, *37* 445, 1952

Jucker, P Die Diagnose der primaren bosartigen Geschwulste des Perikards und der das Perikard infiltrierenden Herzwandtumoren Ztschr f klin Med, *139* 208, 1941

K

Kahlstorf, A Ueber eine orthodiagraphische Herzvolumenbestimmung Fortschr a d Geb d Rontgenstrahlen, *45* 123, 1932

Kahlstorf, A Das Verhalten der Herzgrosse bei Herzjagen Klin Wchnschr, *2* 1028, 1936

Kahlstorf, A, and Ude Die Aenderungen von Herzvolumen und Schlagvolumen nach korperlicher Arbeit Ztschr f klin Med, *125* 85, 1933

Kalisch, Z Ueber einen radioskopisch diagnostizierten und autoptisch bestatigten Fall von partiellem Herzaneurysma Wien klin Wchnschr, *40* 1078, 1927

Kamenetsky, P M, and Rabinowitsch, J S Zur Symptomatologie der Pericarditis exsudativa. Munchen med Wchnschr, *73* 478, 1926

Kappeli, A Ueber einen Fall von Aneurysma der Pulmonalarterie Ztschr f klin Med, *123* 749, 1933

Kaufmann, L Zur Frage der Aorta angusta Ein Beitrag zu den Normalmassen des Aortensystems Veroff Kreigs- u Konstit path, 1919, H 2

Kaufmann, R Ueber Herzerweiterungen Wien Arch f inn Med, *1* 211, 1920

Kaufmann, R, and Meyer, H H Ueber therapeutische Herzverkleinerungen Wien klin Wchnschr, *13* 1154, 1917

Kaufmann, R, and Schwarz, G Einstellvorrichtung fur Herzfernaufnahmen Wien klin. Wchnschr, *30* 889, 1917

Kaunitz, H Hydroperikard bei inkretorischer Storung Ztschr f. klin Med, *130* 601, 1936

Kaup, J Untersuchungen uber die Norm Munchen med Wchschr, *69* 189, 1922

Kautz, F G , and Pinner, M Extrapericardial fat bodies Am J Roentgenol., *35* 40, 1936

Kautsky, A. Ein Fall von diffuser Dilatation der Art. pulmonalis bei congenitaler Insuffizienz der Pulmonalklappen Rontgenpraxis, *8* 809, 1938

Key, E , and Åkerlund, A Verkalktes Aneurysma in der Art renalis. Fortschr. a.d Geb d. Rontgenstrahlen, *25* 551, 556, 1917/18

Kienbock, R Ueber die intrathorakale Struma Med Klin , *4* 488, 1908

Kienbock, R Zur Differentialdiagnose zwischen Aortenaneurysma und Mediastinaltumor Verh dtsch Ges inn Med , *37* 265, 1914

Kienbock, R Lokalisation von Geschossen im Brustkorb Fortschr a d Geb d Rontgenstrahlen, *25* 263, 1917/18

Kienbock, R Radiologie des Herzens (anatomische Einteilung der Herzfigur). Ztschr f klin Med , *86* 64, 1918

Kienbock, R Geschosse im Herzen bei Soldaten Dtsch Arch. f klin. Med , *124* 419, 1918.

Kienbock, R Zur rontgenologischen Differentialdiagnose von Aortenaneurysma und Mediastinaltumor. Fortschr a d Geb d Rontgenstrahlen, *34* 849, 1926.

Kienbock, R. Zur Differentialdiagnose der rechtseitigen extrakardialen Sinusaneurysmen der Aorta und der abgesackten cystischen Perikardialexsudate. Wien med. Wchnschr , *77* 558, 1927

Kienbock, R Zystoide Massen im Brustkorb Fortschr a d Geb d Rontgenstrahlen, *45* 308, 1932

Kienbock, R Ueber das dissezierende Aneurysma der Aorta Fortschr a d Geb d Rontgenstrahlen, *59* 494, 1939

Kienbock, R , Selig, A , and Beck, R Untersuchungen an Schwimmern. Munchen. med Wchnschr., *54* 1427, 1486, 1907

Kienbock, R , and Weiss, K. Fall von grossem Aneurysma dissecans des Aortenbogens Rontgenbefund Wien Ges f inn Med , Sitzg, 28, V, 1925

Kienbock, R., and Weiss, K Ueber entzundlichen Perikard-Divertikel Fortschr a d Geb d Rontgenstrahlen, *40* 389, 1929

Kienbock, R , and Weiss, K Ueber die entzundliche Herzbeuteldivertikel Fortschr a d Geb d. Rontgenstrahlen, *50* 442, 1934

Kirch, E Der Einfluss der linksseitigen Herzhypertrophie auf das rechte Herz Beitr z path Anat u z allg Path , *73* 35, 1924

Kirch, E Der Entwicklungsablauf der rechtseitigen tonogenen Herzdilatation bei Mensch und Versuchtier und seine physiologischer Erklarung Arch f path Anat , *291* 683, 1933

Kirch, E Ueber tierexperimentelle Erzeugung von tonogener Dilatation und Hypertrophie des rechten Herzens durch hochdosierte Histamininjektionen Arch f exper. Path , *171* 691, 1933

Kirch, E Entgegnungen auf die vorstehenden Ausfuhrungen von A Ruhl "Ueber Herzinsuffizienz durch Histamin " Arch. exper Path , *174* 758, 1934.

Kirch, E Anatomische Grundlagen des Sportherzens Verh dtsch Ges inn. Med , *47* 73, 1935

im Tierversuch Arch Kreislaufforsch , *4* 1, 1939

Kirchhof, H , and Lauer-Schmaltz, H Klinische, rontgenologische und kymographische Untersuchungen bei einem Fall von Aortenisthmusstenose. Ztschr f Kreislaufforsch , *35* 135, 1943

Kirsch, O Grundlagen der orthodiagraphischen Herzgrosse und Thoraxbreitenbeurteilung im Kindesalter Berlin, Karger, 1929

Kirsch, O Welche Art der Relativitat ist der Beurteilung der relativen Herzgrosse zugrunde zu legen? Jahrb Kinderh , *137* 185, 1932

Kirschmann, K Herzaneurysma. Fortschr a d Geb. d Rontgentrahlen, *36* 668, 1927.

Kissen, M. Pulmonary insufficiency with a supernumerary cusp in the pulmonary valve Am Heart J , *12* 206, 1936

pregnancy and the puerperium Acta med Scandinav , *138* 421, 1950

Kjellberg, S. R and Ruhde, U. Electrokymographic studies of coarctation of the aorta Acta radiol., *34* 145, 1950

Klaffen, E., and Palugyay, J Vergleichende Untersuchungen uber Lage und Ausdehnung von Herz und Lunge in der Schwangerschaft und im Wochenbett Arch f. Gynak, *131* 347, 1927.

Klason, T · Pericarditis calculosa und Herzverkalkungen. Acta radiol, *1* 162, 1921/22

Klason, T. On the horizontal orthoprojection of the heart Acta radiol, *11* 57, 1930

Klaus, E. J. Untersuchungen zur Klarung eines plotzlichen Todesfalls beim Wettschwimmen Dtsch. Arch f klin Med, *181* 285, 1937

Klaus, E J Untersuchungen uber die Ruckbildung der Ueberanstrengungsdilatation des Herzen bei einem jugendl Wettkampfsportler Ztschr f Kreislaufforsch, *31* 7, 1939.

Klaus, E J, and Albert, A H Die morphologischen und funktionellen Herzveränderungen in dosierten Valsalvaschen Versuch bei jugendl. Wettkampfsportlern. Dtsch Arch f. klin Med, *182* 477, 1938, *183* 315, 1938.

Kleemann, M Ueber den Wert der Zahlen in der Orthodiagraphie. Dtsch med Wchnschr, *45* 621, 1919

Kleinschmidt, K, and Wachsmuth, W Experimentelle Untersuchungen an Herz und Kreislauf bei arteriovenoser Fistel Dtsch Ztschr f. Chir, *201* 145, 1927.

Klemola, E Ueber Isthmusstenose der Aorta Duodecim 53 1139, 1937

Klinck, G H, and Hunt, H D Pulmonary varix with spontaneous rupture and death Arch Path, *15* 227, 1933

Kloiber, H, and Hochschild, H Zur Frage des rontgenologischen Sichtbarwerdens des Herzens im Perikardialerguss Fortschr a d Geb d Rontgenstrahlen, *27* 473, 1919/21.

Kment, H Aneurysma der Arteria renalis. Beitr z klin Chir, *147* 144, 1929

Knipping, H. W Die verschiedenen Formen des Lungenodems und ihre Behandlung. Therap. d. Gegenw, *76* 385, 1935

Knothe, W Die "Hiatushernien" von Standpunkt des Rontgenologen Dtsch med Wchnschr., *58* 609, 1932

Knox, R The investigations on the movements of the heart by the use of slit diaphragm and the moving film Brit J Radiol, *21* 142, 1925

Koch, W Ueber den Verschluss der Koronararterien Med Klin, *26* 1139, 1930.

Koch, W, and Wieck, W Anatomische Analyse des Rontgenbildschattens des Herzen und der Interlobarspalten der Lunge Jena, Fischer, 1930

Koepplin, F Zur Morphologie und Funktion des Sportherzens Schweiz med Wchnschr, *39* 1053, 1950

Kohlmann, G Die Klinik und Rontgendiagnose des Lungeninfarkts Fortschr a d Geb d Rontgenstrahlen, *32* 124, 1924

Kohler, A Die Herstellung fast orthodiagraphischer Herzphotogramme mittels Rontgeninstrumentarien mit kleiner Elektrizitatquelle Wien klin Rundschau, *19* 279, 1905

Kohler, A Telerontgenographie des Herzens Dtsch med Wchnschr, *34* 186, 1908

Kohler, A Rontgenographischer Nachweis von Kalkplatten im Aortenbogen Lebender Fortschr a d Geb d Rontgenstrahlen, *18* 194, 1912

Kohn, H Angina pectoris und Unfall Klin Wchnschr, *8* 795, 1929

Kommerell, B Verlagerung des Oesophagus durch eine abnorm verlaufende Arteria subclavia dextra (Arteria lusoria) Fortschr d Geb d. Rontgenstrahlen, *54* 590, 1936

Kommerell, B Verkalkte Herzklappen im Rontgenbild Fortschr a d Geb d Röntgenstrahlen, *53* 34, 1936

[illegible]

Koppenstein, E [illegible] Fortschr. a d. Geb d Rontgenstrahlen, *36* 139, 1927.

Kornblum, K, Bellet, S, and Ostrum, H W.· Tuberculous pericarditis. Its roentgenological significance Am J Roentgenol, *29* 202, 1933.

Korner, G Kurze Mitteilung einer Aortenmissbildung (Hohe Recthlage mit Verlagerung des Oesophagus und der Trachea) Fortschr a d. Geb. d Rontgenstrahlen, *52* 400, 1935.

Kottenhoff Luft f med, 1938, 2.

Kovacs, F, and Stoerck, O. Ueber das Verhalten des Oesophagus bei Herzvergrosserung Wien klin. Wchnschr, *23* 1471, 1910

Kratzeisen, E. Allgemeine Herzvergrosserung nach Diphtherie. *Zentralbl f. Herz u. Gefasskrankh* *12* 185, 1920

Kraus, F Ueber das Kropfherz. Wien klin. Wchnschr., *49* 416, 1899

Kraus, F Einiges uber funktionelle Herzdiagnostik Dtsch med Wchnschr., *31* 1, 52, 90, 1905.

Kraus, F Konstitutionelle Herzschwache. Med Klin, *1* 1271, 1905.

Kraus, F Ueber Kropfherz Dtsch med Wchnschr, *32* 1889, 1906

Kraus, F. Ueber die Aortenerweiterung bei der Dohle-Hellerschen Aortitis Dtsch med. Wchnschr, *40* 377, 1914

Kraus, F. Ueber sogenannte idiopathische Herzhypertrophie Berl. klin Wchnschr, *54* 765, 1917 Med Klin, *13* 926, 1917

Kraus, F Ueber konstitutionelle Schwache des Herzens Dtsch. med Wchnschr., *43* 1153, 1917.

Kraus, F *Ueber die Moglichkeit der klinische Diagnose intrakardialer Aneurysmen Berl klin* Wchnschr, *56* 529, 1919

Kreuzfuchs, S Ein neues Verfahren der Herzmessung. Munchen med Wchnschr., *59* 1030, 1912.

Kreuzfuchs, S Die Brustaorta im Rontgenbilde Wien klin Wchnschr, *29* 701, 1916

Kreuzfuchs, S Ein typischer Aortenbefund von prinzipieller Bedeutung bei Pleuritis mediastinalis superior dextra Wien med Wchnschr, *85* 871, 1935

Kreuzfuchs, S Aortenisthmusmessung mittels transparenter Kreise Med Klin, *31* 1274, 1935.

Kreuzfuchs, S Aortenverlauf und Messbarkeit im Kindesalter Fortschr a d Geb d Rontgenstrahlen, *54* 396, 1936

Kreuzfuchs, S Okkult-pulmonale Kardiopathie Ztschr f Kreislaufforsch, *28* 841, 1936

Kreuzfuchs, S Altersbestimmung an Aortenrontgenogrammen von Lebenden und post mortem *vermittels einer geometrischen Aortenmessmethode Wien klin Wchnchr*, *48* 1355, 1935.

Kreuzfuchs, S Die einfachste Aortenmessung und ihre physiologische klinische Bedeutung Munchen med Wchnschr, *83* 681, 1936

Kreuzfuchs, S Pulmonalismessung Fortschr a d Geb d Rontgenstrahlen, *56* 756, 1937.

Krohn, K H Rippenusuren bei einen Fall von kongenitaler Isthmusstenose Rontgenpraxis, *9* 776, 1937

Kronenberger, F, and Leeser, F Isolierter Dilatation des rechten Vorhofes Rontgenpraxis, *2* 924, 1930

Kudisch, B M Rongtenologische Studien der Aorta I Mitteilung Fortschr a d Geb d Rontgenstrahlen, *48* 298, 1933

Kudisch, B M Ueber die funktionell-dynamische Methodik der Kardiorontgenographie Fortschr d Geb d Rontgenstrahlen, *46* 529, 1932.

[illegible] of two cases of truncus [illegible] 280, 1932

Kuhlmann, F Klinische Bedeutung der Rontgendiagnostik der Coronarsklerose Fortschr. a d Geb d Rontgenstrahlen, *48* 42, 1933

Kuhlmann, F Zur Rontgendiagnostik der Coronarthrombose. Klin Wchnschr., *13* 957, 1934

Kulbs, F Experimentelles uber Herzmuskel und Arbeit Arch exper Path. Pharm, *53* 288, 1906

Kulbs, F Ueber den Einfluss der Bewegung auf den wachsenden und erwachsenen Organismus Dtsch med Wchnschr, *38* 1916, 1912

Kukula, O Beitrag zur Kasuistik und operativen Behandlung von Herzsteckschussen Med Klin, *13* 907, 1917

Kung, S. K. Trifft die Spitzersche Theorie fur die Falle von Transposition der grossen Gefasse zu? Beitr z path Anat u z allg Path, *88* 127, 1931.

L

Lam, C Pericardial coelomic cyst. Radiology, *48* 239, 1947

Lambert, A. V. Etiology of thin-walled thoracic cysts J Thoracic Surg, *10* 1, 1940

Lange, F., and Wehner, E Das Herz bei Hypertonie und bei Arteriosklerose. Dtsch Arch f klin Med, *160* 45, 1928

Lange, R., and Feldmann, H. Herzgrössenverhaltnis bei Rontgendurchleuchtung. Monatschr. f. Kinderh., 21 458, 1921.

Laplace, L. B. Observations on the effect of an arteriovenous fistula on the human circulation. Am. J. M. Sc., 189 497, 1935.

Lasius, O. J. Vorubergehende Herz- und Aortendilatation und Vestibularisstorung bei einem 29 jahrigen Elektromonteur, verursacht durch Starkstromunfall von 50,000 Volt. Med. Klin., 25 1133, 1929.

Lauber, H., Przywara, E. L., and Velde, G. Zur Messung des Aortendurchmessers in bestimmter pulsationsphase. Beitrag zur Schlagvolumbestimmung auf physikalischem Wege. Ztschr. f. klin. Med., 119 67, 1931.

Laubry, C. Sur le diagnostic radioscopique des anévrismes de l'aorte abdominale. Bull. d. l. Soc. d. méd. d. hôp. de Paris, 44 1293, 1920.

Laubry, C., Cottenot, P., Routier, D., and Heim de Balsac, R. Étude anatomoradiologique du coeur et des gros vaisseaux par opacification. J. Radiol. et Électrol., 19 195, 561, 700, 1935, 20 65, 1936.

Laubry, C., and Heim de Balsac, R. Mise en evidence par la radiokymographie de la sténose isthmiques. Arch. d. mal. du coeur, 30 394, 1937. Valeur des erosions costales dans le diagnostic de sténose isthmiques. Ibid., 30 128, 1937.

Laubry, C., and Pezzi, C. Traité des maladies congenitales du coeur, Paris, 1921.

Laubry, C., Souhé, P., and Heim de Balsac, R. Le syndrome phrenogastrique des coronarites. Arch. d. mal. du coeur, 31 583, 1938.

Laubry, C., Routier, D., and Heim de Balsac, R. Grosse pulmonaire. Petite aorta. Affection congenitale. Bull. d. l. Soc. d. med. d. hôp. de Paris, 56 847, 1941.

Laubry, C., and Thomas, M. Les lesion de l'artère pulmonaire et leurs conséquences au cours du retrecissement mitral. Bull. d. l. Soc. d. méd. d. hôp. de Paris, 50 639, 1926.

Lauenstein, H. Ueber kardial bedingte Bronchostenosen und Lungenatelektasen im Kindesalter. Ztschr. f. Kinderh., 54 145, 1933.

Laurell, H. Ueber respiratorische Veranderungen im Lungfeld, Mediastinum, und Zwerchfell unter normaler Verhaltnissen und bei gewissen krankhaften Zustanden der Lunge und des Brustfells. Acta radiol., 8 55?, 1927.

Laurell, H. Rontgenologische Herzstudien. Festschr. f. Quensel. Upsala lakareforening, 34 495, 1928.

Laurell, H. Die orthostatic arterielle Anamie, ein gewohnliches, aber oft fehlgedeutetes Krankheitbild. Fortschr. a. d. Geb. d. Rontgenstrahlen, 53 501, 1936.

Ledoux-Lebard, R. Balle de shrapnell libre dans l'oreillette droite. J. Radiol., 2 34, 1916.

Ledoux-Lebard, R., Calderon, J. G., and Eliopoulos, S. Le radiodiagnostic des calcifications de l'aorte abdominale. Bull. Acad. Méd. Paris, 109 407, 1933.

Ledoux-Lebard, R., Garcia-Calderon, J., and Ledoux-Lebard, G. Le radiodiagnostic des calcifications de l'aorte abdominale et de ses branches. Presse méd. 43 2084, 1935.

Leendertz, G. Warum sind Stauungsergusse in der Pleurahohle rechts haufiger als links? Dtsch. med. Wchnschr., 67 348, 1941.

Leeser, F. Der Lobus venae azygos im Rontgenbilde (mit einem pathologisch-anatomisch kontrollierten Fall). Rontgenpraxis, 1 267, 1929.

Lehmkuhl, H. Beitrag zur Rontgenologie des kindlichen Herzens. Fortschr. a. d. Geb. d. Rontgenstrahlen, 38 128, 1921.

Lemke, H. H. Gibt es eine fur Insuffizienz der Segelklappen typische Vorhofpulsation? Fortschr. a. d. Geb. d. Rontgenstrahlen, 59 372, 1939.

Lenk, R. Zur Rontgendiagnostik der Aneurysmen der Aorta descendens und der Aortenlues uberhaupt. Fortschr. d. Geb. d. Rontgenstrahlen, 30 135, 1922/23.

Lenk, R. Rontgendiagnose der Koronarsklerose in vivo. Gleichzeitig ein Beitrag zur Erkennbarkeit des Herzaneurysmas im Rontgenbilde. Fortschr. a. d. Geb. d. Röntgenstrahlen, 35 1265, 1927.

Lenk, R. Die Rontgendiagnostik der intrathorakalen Tumoren und ihre Differentialdiagnose. Wien, Springer, 1929.

Lerman, J., Clark, R. J., and Means, J. H. The heart in myxedema. Ann. Int. Med., 6 1251, 1933.

Levene, G., and Kaufman, S A. The roentgen diagnosis of pericardial effusion, with special reference to the appearance of the barium-filled esophagus and the cardiohepatic angle Radiology, *57* 373, 1951

Levy, L, Fowler, R, Jacobs, H, Leckert, J, Irion, J, Rosen, J, and Chastant, H. Angiocardiographic confirmation of pericarial effusion Am Heart J, *43* 59, 1952

Levy-Dorn, M Zur Beurteilung des Herzgrosse Berl klin. Wchnschr, *53* 624, 1916

Le Wald, L T, and Turell, G H The aviator's heart Roentgen ray studies under conditions simulating high altitudes Am. J Roentgenol, 7 67, 1920.

Lewis, T Material relating to coarctation of the aorta of the adult type. Heart, *16* 205, 1933

Lewis, T, and Drury, A W Observations relating to arteriovenous aneurysm Heart, *10* 301, 1923

Lian, C, Marchal, M, and Deparis, M. Le diagnostic clinique et radiologique des anévrismes aortiques intrapéricardiques (anévrismes des sinus de Valsalva). Bull Soc méd d. hôp de Paris, *56* 522, 1933

Lian, C, and Marchal, M L'examen radiologique de l'aorte en position frontale Presse méd, *1* 841, 1936

Lichtman, S S Isolated congenital dextrocardia Arch Int Med, *48* 866, 1931

Lillie, W J, McDonald, J R., and Clagett, O T Pericardial coelomic cysts and pericardial diverticula J Thoracic Surg, *20 409, 1950*

Lind, J, and Wegelius, C Angiocardiographic studies in children Advances in pediatrics, New York, Academic Press, 1952, *5*.

Lindboe, E F Aneurysm of the splenic artery diagnosed by x-rays and operated upon with success. Acta chir Scandin, *72* 108, 1932

Lindgren, E Roentgendiagnosis of arterio-venous aneurysm of the lung Acta radiol, *27*·585, 1946.

Lippmann, A, and Quiring, W Die Rontgenuntersuchung der Aortenkrankungen mit spezieller Berucksichtigung der Aortenlues Fortschr a d Geb d Rontgenstrahlen, *19* 103, 1912/13

Ljungdahl, M Ein Fall von Pneumoperikardium, Dtsch Arch klin Med, *111* 19, 1913

Loben, F Ueber angeborene Rechtlagerung des Herzens bei normalem Situs der Bauchorgane Fortschr a d Geb d. Rontgenstrahlen, *38* 553, 1928

Lochte Beitrag zur Kenntnis des Situs transversus partialis und der angeborenen Dextrocardie. Beitr z path Anat, *16* 61, 1894

Lochte Ein Fall von Situs viscerum irregularis nebst einem Beitrag zur Lehre von der Transposition der arteriellen grossen Gefassstamme des Herzens Beitr. z path Anat., *24* 124, 1898

Loewy, A, and Mayer, A E Ueber experimentell erzeugte akute Herzerweiterung beim Menschen Klin Wchnschr., *5* 1213, 1926

Lohmann, C W Zur Röntgensymptomatologie des Morbus coeruleus Fortschr. a d. Geb d. *Rontgenstrahlen, 52 43, 1935.*

Lopo de Carvalho and Egas Moniz Visibility of the pulmonary vessels Angiopneumographie Acta radiol, *14* 433, 1933

Loweneck, M Einige seltene Beobachtungen aus de Oesophaguspathologie Dysphagia lusoria bei hoher Rechtslage der Aorta Fortschr a d Geb d. Röntgenstrahlen, *35* 1230, 1927.

Ludin, M. Aneurysma der Arteria pulmonalis Acta radiol, *14* 259, 1933.

Ludke, H, and Schuller, L. Ueber die Wirkung experimenteller Anamie auf den Herzgrosse. Dtsch Arch f. klin Med., *100* 512, 1910

Ludwig, H Funktionelle Mitralstenosen durch Tumoren des linken Vorhofs Ztschr f klin Med, *123* 587, 1933

[illegible] Fortschr a d Geb d Rontgen-

[illegible] des Herzens wahrend bestimmter Aktionsphasen Fortschr. a d.

[illegible] Rontgenstrahlen,

Luger, A. Zur Symptomatologie der Insuffizienz der Aortenklappen mit beonderer Berucksichtigung der Frage der relativen Insuffizienz derselben Wien med Wchnschr, *78* 209, 1928

Luisada, A Aneurisma vero dell'arterie polmonare da arterite luetica Minerva med, *25* 421, 1934.

Luisada, A A, and Fleischner, F G Studies of fluorocardiography, tracings of the left ventricle in myocardial infarction. Acta cardiol (Bruxelles) *4* 179, 1948.

Luisada, A. A and Fleischner, F. G Dynamics of the left auricle in mitral valve lesions Am J Med, *4* 791, 1948

Luisada, A A, Fleischner, F G, and Rappaport, M. B Fluorocardiography (Electrokymography) Am Heart J, *35* 336, 348, 1948

Lutembacher, R De la sténose mitrale avec communication interauriculaire Arch d mal du coeur, *9* 235, 1916

Lutembacher, R Anévrisme de l'oreilette gauche Arch d mal du coeur, *10* 145, 1917.

Lutembacher, R Deux nouveaux cas d'anévrisme de l'oreilette gauche Arch d mal du coeur, *11* 434, 1918

Lutembacher, R Anévrismes du ventricule gauche Étude clinique et radiologue des anévrismes ventriculaires Arch d mal du coeur, *13* 49, 1920

Lutembacher, R Sténose mitral et communication interauriculaire Arch. d mal du coeur, *29* 229, 1936

Luthi, A Ueber Herzschusse Schweiz med. Wchnschr, *56* 454, 1926

Lutz, P Zum Rontgenbild der Vena azygos Zugleich ein Beitrag zur Genese des Lobus venae azygos (Lobus venae cardinalis) Radiol Austriaca *5* 1952

Lysholm, E Rontgenskopischer Modellierungsapparat auch fur Quersektion und Lokalisation Acta radiol, *7* 189, 1926

Lysholm, E, Nylin, G, and Quarna, K The relation between the heart volume and stroke volume under physiological and pathological conditions Acta radiol, *15* 257, 1934

M

Mahaim, J Des variations de l'indice de profondeur dans les réactions volumetriques du coeur Ann Méd, *20* 73, 1926

Mahaim, J De l anévrisme primitif de l'oreillete gauche Troubles particulaires du rhythme cardiaque La dissociation interauriculaire Ann Méd, *21* 380, 1927

Mahaim, J Des réactions volumetriques *du coeur de l'hypertrophie à la dilatation* Ann Méd, *23* 79 1928

Masslich, R, and Bobrezkaja, W Ueber die Grosse des Herzens der Schwangeren Fortschr d Geb d Rontgenstrahlen, *54* 574, 1936

v d Mandele, L J Ueber einen Fall von Pleuritis mediastinalis und uber den Doppelkontur des Herzschattens im Rontgenbild Fortschr a d Geb d Rontgenstrahlen, *34* 84, 1926

Mandelstamm, M, and Reinberg, S Die Dextrocardie Ergebn inn Med, *34* 130, 1928

Mannheimer, F Morbus caeruleus Basel, Karger, 1949

Mannheimer, E, and Nordenfelt, P J Four cases of congenital heart disease Acta pediatr, *23* 200, 1938

Marble, H S, and White, P D Traumatic aneurysm of the right pulmonary artery J A M A, *74* 1778, 1920

Marquis, R M Ventricular septal defect in early childhood Brit Heart J, *12* 265, 1950

Martland, H S Syphilis of the aorta and heart Am Heart J, *6* 1, 1930

Mason, D G Subacute cor pulmonale Arch Int Med, *66* 1221, 1940

Master, A M Roentgenoscopy as a diagnostic aid in coronary occlusion Am J Roentgenol, *45* 350, 1941

Master, A M Right sided aorta with atypical coarctation involving only the left subclavian artery Am Heart J, *32* 778, 1946

Mauthner H, and Lowy Transposition der Aorta oder Persistenz einer rechtkammerigen Aorta Arch f path Anat, *229* 337, 1921

Mazer, M L True pericardial diverticula J Thoracic Surg, *55* 27, 1950

McCartney J I and Drummond, H A case of gunshot wound of the liver where the missile,

entering the circulation through the hepatic vein, was found lodged in the right ventricle of the heart Brit J Surg, 5 508, 1918.

McClure, C C. A case of aneurysm of the abdominal aorta. Radiology, *17* 825, 1931.

McCrea, F D, Lyster, J A, and Meek, W. J The effect of exercise upon diastolic heart size. Am J Physiol, *83* 361, 1927

McGinn, S, and White, P D Interauricular septal defect associated with mitral stenosis. Am Heart J, *9* 1, 1933.

McGinn, S, and White, P D Epipericardial fat, its non-recognition as a common cause of error in x-ray measurement of heart size J A M A, *107* 200, 1936

McKusick, V A Chronic constrictive pericarditis II Electrokymographic studies and correlations with roentgenography, phonocardiography and right ventricular pressure curves. Bull Johns Hopkins Hosp, *90* 27, 1952

McPhedran, F M, and Weyl, C N Automatic synchronization of roentgen ray exposures Am J M Sc, *169* 510, 1925.

Mead, K C Persistent patency of the ductus arteriosus J A M A, *55* 2205, 1910

Meek, W J The effect of changes in pulse rate on diastolic heart size Am J Physiol, *70* 385, 1925

Meek, W J, and Eyster, J A The effect of plethora and variations in venous pressure on diastolic size and output of the heart Am J. Physiol, *61* 186, 1922

Meister, W, and Derlich, H Rontgenologische Herzgrossenbestimmungen beim Menschen im Unterdruck Luft-med, *3* 32, 1939

Melchart, F Herzneurysma, in vivo diagnostiziert und autopisch bestatigt Rontgenpraxis, *1* 887, 1929

Melchoir, E Zur Kenntnis der Herzstorungen bei arterio-venosen Aneurysmen und ihre Heilbarkeit durch Operation Med Klin, *25* 514, 1929

Meteanu, C, Dubost, C, Durand, M, and Hoffmann, T Bemerkungen zur klinischen Diagnose und zur operativen Behandlung kongenitaler Herzfehler auf Grund von 1000 untersuchten und 450 operierten Fallen Arch f Kreislaufforsch, *18* 21, 1952

Metzger, H N, and Ostrum, H Right sided aortic arch Am J Digest Dis and Nutrition, *6* 32, 1939

Meyer, A W, and Sulger, E Das Kropfherz vor und nach der Operation Med Klin. *22* 834, 1926.

Meyer, L Zur Kenntnis des kleinen Herzens Dtsch med Wchnschr, *46* 789, 1920, *49* 1384, 1923

Meyer, E. Ueber Herzgrosse und Blutgefassfullung Klin Wchnschr., *1* 1, 1922

Meyer, E, and Seyderhelm, R Beziehungen zwischen Herzgrosse und Blutzusammensetzung. Verh dtsch Kongr inn Med, *32* 376, 1921

Meyer, P Dextrocardie pure congénitale sans inversions des cavités cardiaques Arch d mal. du coeur, *30* 971, 1937

Meyer-Borstel, H Ueber Form und Grossenveranderungen des Herzens bei Struma Fortschr. a d Geb d Rontgenstrahlen, *41* 695, 1930.

Micks, R H Congenital aneurysms of all three sinuses of Valsalva. Brit Heart J, *2* 63, 1940

Miller, M K, and Lyon, W W Persistent truncus arteriosus cardiac hypertrophy, dysphagia, death on eleventh day Am Heart J, *7* 106, 1931.

Misske, B, and Schone, G Das Herz im Rontgenbilde bei Schilddrusenuberfunktion Fortschr. a d Geb d Röntgenstrahlen, *50* 121, 1934

Moberg, G Some views on the important part played by the pressure conditions in chest and abdomen for the radiographic examination of heart and lung Acta radiol, *21* 1, 1940

Molnar, J Ueber Luftansammlung in Herzbeutel. Fortschr a d Geb. d Rontgenstrahlen, *36* 656, 1927.

Monckeberg, J. G Isthmusstenose der Aorta Handbuch der speciellen pathologischen Anatomie, Bd II, S. 105.

Moreau, R, and Boudin, G Kystes hydatiques multiples du coeur Arch d mal du coeur, 34 142, 1941.

Moritz, F. Ueber Veranderungen in der Form, Grosse und Lage des Herzens beim Uebergang aus horizontaler in vertikale Korperstellung usw. Dtsch Arch f klin. Med, 82 1, 1905.

Moritz, F. Ueber funktionelle Verkleinerung des Herzens Munchen med Wchnschr, 55 713, 1908

Moritz, F. Demonstration zur Grosse und Fassungskraft des Herzens Verh dtsch Ges Kreislaufforsch, 1 125, 1928

Moritz, F.. Ueber die Norm der Grosse und Form des Herzens beim Manne. Dtsch Arch klin. Med, 171 431, 1931, 172 462, 1932, 174 330, 1932

Moritz, F Grosse und Form des Herzens bei Meistern im Sport Dtsch Arch f klin Med, 176 455, 1934

Moritz, F Herzdilatation München med Wchnschr, 82 450, 1935

Moritz, F. Zur Beurteilung der Herzgrosse Fortschr d a Geb d Rontgenstrahlen, 38 993, 1928

Moritz, F, and Mohr, W Ueber die Kapazitat und die Muskelmasse der menschlichen Herzhohlen Ztschr f Kreislaufforsch, 25 609, 1933

Mosler, D, and Balsanoff Ueber den Valsalva-Versuch Klin Wchnschr, 3 401, 1924

Muir, D C, and Brown, J W Patent intraventricular septum (maladie de Roger). Arch Dis. Childhood, 9 27, 1934

Muller, E F Perikarditische Verkalkungen, Greifswald, 1918

Muller, H, Jr Zur klinischen und pathologischen Anatomie des unkomplizierten offenen Septum ventriculorum Dtsch Arch f klin Med, 133 316, 1920

Müller, H, Jr Die kongenitale Aortenkonusstenose. Schweiz med. Wchnschr, 54 702, 1924

Muller, H, Jr. Die unkomplizierte angeborene Pulmonalstenose. Schweiz med Wchnschr, 55 619, 1925.

Muller, H, Jr. Vorhofseptumdefekt ohne weitere Herzmissbildung Ein Beitrag zu den gutartigen angeborenen Herzfehlern Schweiz. med Wchnschr, 57 862, 1927

Müller, H Jr Ein Sektionsfall einer unkomplizierten angeborenen Aortenstenose Schweiz. med. Wchnschr, 62 162, 1932

Muller, K Ein Fall von Isthmusstenose Wien Ges f Rontgenkd, April, 1934

Müller, W. Die Massenverhaltnisse des menschlichen Herzens. Hamburg Voss, 1883

Muller, W Fall von multilokularen Dermoiden des Mediastinums. Sitzg d Wien Rontgenges, Mai, 1948

N

Nagel, M Beitrage zur Kasuistik und Lehre von der angeborenen reinen Dextrokardie Dtsch. Arch f. klin med, 96 552, 1909

Natvig, P The volume of the heart in Muller's and Valsalva's tests Acta radiol., 15 657, 1934

Nauwerck, C Lungenvarix und Hamoptoe Munchen med Wchnschr, 70 1084, 1923.

Nauwerck, C Varixknoten im Herzen Centralbl f allg. Path, 32 113, 1920/21.

Neiman, B H Varix of the pulmonary vein Am. J Roentgenol, 32 608, 1934

Nemenow, M Rontgendiagnostik der Aneurysmen der Bauchaorta Pneumoperitoneum. Fortschr. a d Geb d Rontgenstrahlen, 37 641, 1928.

Német, G Zur Kenntnis der "Mitralform" gesunder Herz. Klin Wchnschr., 2 348, 1923.

Német, G, and Schwedel, J Roentgenographic studies of the right ventricle Am Heart J, 7 132, 1932

Neuburger, J Zwei Falle von syphilitischen Aneurysmen der Aorta pulmonalis Dtsch. med Wchnschr, 56 821, 1930

Nicolai, G F, and Zuntz, N Fullung und Entleerung des Herzens bei Ruhe und Arbeit. Berl. klin Wchnschr., 51 821, 1941.

Nolte, F. A Ueber der Veranderung der Herzform und -grösse unter der Einwirkung intrapulmonaler [illegible] gramm [illegible]

Nordenfelt, O [illegible] Acta med Scandinav, 93 297, 1937

Nylin, G The relation between heart volume and stroke volume in recumbent and erect position Skand Arch Physiol, *69* 732, 1934

O

Odquist, H A roentgen cinematographic study of the movements of the mitral ring during heart action Acta radiol, *26* 392, 1945

Oheim, L Herzmuskelveranderungen bei Diphtherie, ihre zeitliche Aufeinanderfolge und topographische Verteilung Beitr z path Anat. u z allg Path, *100* 195, 1938.

Ohler, W R, and Abramson, J The heart in myxedema Arch Int. Med, *53* 165, 1934

O'Kane, A, Andrew, F D, and Warren, A A standardisation roentgenologic study of the heart and great vessels in the left oblique view Am J Roentgenol, *23* 373, 1930

Oppenheimer, B S Idiopathic dilatation of the pulmonary artery Cited after Taussig

Oppler, B, and Sielmann, H Ein Beitrag zur Diagnose der Aorta descendens-Erkrankungen Munchen med Wehnschr, *75* 954, 1928

v. Oppolzer, R Zwei Falle von Aneurysma arteriovenosum Wien klin Wehnschr, *58* 246, 1946

Osgood, F F, Gourley, M F, and Baker, R L Diagnosis of dissecting aneurysm of the aorta Ann Int Med, *9* 1398, 1936.

Otten, M Die Bedeutung der Orthodiagraphie fur die Erkennung der beginnenden Herzerweiterung Dtsch Arch f klin Med, *105* 370, 1912

Ottonello Bemerkungen zur normalen Rontgenanatomie des Thorax Fortschr. a d Geb d. Rontgenstrahlen, *45* 677, 1932

P

Packard, M., and Wechsler, H Aneurysm of the coronary arteries Arch Int. Med, *43* 1, 1929

Pal, J Die Tonuskrankheiten des Herzens und der Gefasse Wien, Springer, 1924

Palmieri, G. G Ortodiagrafia e cardiovolumetria Gior clin med, *1* 146, 1920

Palmieri, G. G. Sul valore della ricerca dell angolo di scomparsa della punta (metodo Vaquez-*Bordet) come indice del volume a relativo cuore Variazioni di tale angolo nello stesso individuo* e loro cause Mal Cuore, *4* 184, 1920

Palmieri, G G Ueber meine Methode der plastischen Darstellung des Herzens am Lebenden. Acta radiol, *10* 127, 1920

Paltauf, R Dextrocardie und Dextroversio cordis Wien klin. Wehnschr, *13* 120, 1901.

Pape, R Ueber einen abnormen Verlauf ("tiefe Rechtslage") des mesaortitischen Aorta descendens *Fortschr a d Geb d Rontgenstrahlen, 46 257, 1932.*

Pape, R Zur Beurteilung des Fettburzels und ahnliches Schatten an der Herzspitze Wien. klin Wehnschr, *59* 285, 1947

Papp, C Le diagnostic radiologique de le tétrade de Fallot. Arch d mal du coeur, *24* 249, 1931

Parade, G W Aneurysmatische Elongation des Herzens Med Klin, *30* 1357, 1934

Parade, G W Basedow und Herz Med Klin, *30* 1398, 1935.

Parade, G W Schilddrusenfunktion und Herz Med Klin, 31 1130, 1935

Parade, G W, and Kuhlmann, F. Zur Rontgendiagnose der Verkalkungen des Herzskeletts Munchen med Wehnschr, *80* 99, 1933

Parade, G. W, and Rahm, H Herzgrosse bei Morbus Basedow. Ztschr. f klin Med, *126* 667, 1934

Parkinson, J Enlargement of the heart Lancet, *1* 1337, 1391, 1936

Parkinson, J, and Bedford, E The pulmonary artery impression on the oesophageus Lancet, 2:337, 1931

Parkinson, J, and Bedford, E The aortic triangle. Lancet, *2* 909, 1936

Parkinson, J, and Cookson, H The size and shape of the heart in goitre Quart J Med, *24* 499, 1931

Passler, H Ueber Ursache und Bedeutung der Herzaffektion Nierenkranker Sammlg klin Vortr, *1906*, Nr 408 (Int Med, Nr 123)

... effect of exercise on the human heart in

... myomatose des Herzens bei Geschwistern

Peiffer, H Der Kreislauf unter Beschleunigung Luftf med, *3* 32, 1939
Pernkopf, E. Topographische Anatomie Wien Urban und Schwarzenberg, 1938
Pernkopf, E, and Wirtinger, W Das Wesen der Transposition im Gebiete des Herzens, ein Versuch der Erklarung auf entwicklungsgeschichtlicher Grundlage. Arch f path Anat, *295* 143, 1935
Pezzi, F, and Agostino, G Considérations cliniques, anatomiques et radiologiques, à propos d'un cas de maladie congénitale du coeur controlée à l'autopsie, persistance du tronc artérial commun Arch d mal du coeur, *21* 117, 1924
Pfeiffer, R Das Verhalten der Herzlage nach Phrenicusexarese Med Klin, *31* 813, 1935.
Pfeiffer, W Ueber der Veranderung der Lungenzeichnung bei Atmungsproben und deren diagnostiche Verwertbarkeit Dtsch Arch f klin Med, *180* 266, 1937
Pfeiffer, W Herzkfunktionsprufung im Rontgenbild Fortschr a d Geb d Rontgenstrahlen, *58* 547, 1938
Plenge, K Zur Frage der Syphilis der Lungenschlagader Arch f path Anat, *275* 572, 1930
Pletnew, D D Lasst sich ein Aneurysma der Herzventrikel intra vitam feststellen? Ztschr f. klin Med, *104*, 378, 1926
Podkaminsky, N A Beitrage zur pathologischen Arbeitsphysiologie I Mitteilung Das Herz des Lastragers im Rontgenbilde Arb physiol, *1* 306, 1929
Podkaminsky, N A Entwicklung der Hypertrophie und der Dilatation des Herzens in Abhangigkeit von seinen funktionellen Bau Rontgendiagnostik einter partiellen Hypertrophie und Dilatation Wien klin Wchnschr, *45* 92, 1932
Pohl, R Trichterbrust und Herzform Wien klin Wchnschr, *41* 1439, 1928
Poinso, R L'anévrisme de l'artère pulmonare Gaz d hôp, *100* 477, 1927
Pollitzer, H Ren juvenum Wien, Urban & Schwarzenberg, 1913
Popp, L. Ueber die Herzgeschwulste in Verbindung mit einem Fall von Sarkom den rechten Vorhofohres Fortschr a d Geb d Rontgenstrahlen, *46* 26, 1932
Porter, W B. Heart changes and physiologic adjustment in hookworm anemia Am Heart J, *13* 550, 1937
Porter, W B Diaphragmatic flutter with symptoms of angina pectoris J A M A, *106* 992, 1936
Postel, E, and Laas, E Periarteritis nodosa Ein Bericht uber zwei Falle mit Erkrankung der Lungen Ztschr f Kreislaufforsch, *33* 545, 1941
Potain and Rendu Endarteriitische Wandschadigungen der Pulmonalis bein Endocarditis Cited by Vaquez and Bordet Radiologie du coeur Paris, Bailliere, 1928
Pratje, A Form und Lage der Speiserohre des lebenden Menschen, ein Beitrag zur Topographie des Mediastinums Ztschr Anat u Ent gesch, *81* 269, 1926
Prichard, R W Tumors of the heart Review of the subject and report of one hundred and fifty cases. Arch Path, *51* 98, 1951
Pugh, D G Roentgenologic diagnosis of certain congenital lesions of the heart and great vessels Am J M Sc, *212* 628, 1946
Purks, W K Dynamic dilatation of the thoracic aorta Am Heart J, *9* 655, 1934
Puschel, E Ein Beitrag zur Kenntnis der Herzvergrosserungen im fruhen Kindesalter Arch f Kinderh, *99* 341, 1933
Putschar, W. Glykogenspeicherungskrankheit. Beitr z. path Anat. u z allg Path, *90* 222, 1932

Q

Quaresma, L Geometrische Messung des Aortendurchmessers Fortschr a d Geb d Rontgenstrahlen, *56* 743, 1937
Quattlebaum, J T Arteriovenous aneurysm Am Heart J, *13* 95, 1937.

R

Raab, L Zur Frage von der akuten Dilatation des Herzens durch Ueberanstrengung Munchen. med Wchnschr., *56* 555, 1909.
Raab, W Untersuchungen uber einen Fall von kongenitalem Herzvitium Wien Arch klin med, 7 367, 1923
Raab, W Sudden death of a young athlete Arch. Path, *36* 388, 1943

Raab, W The pathogenetic significance of adrenalin and related substances in the heart muscle Exp Med and Surg, *1* 188, 1943.

Raab, W Sudden death with an excessive myocardial concentration of epinephrine-like substances in a case of obesity and cystic thyroid disease Arch Path, *38* 110, 1944

Railsback, O C, and Dock, W Erosion of the ribs due to stenosis of the isthmus (coarctation) of the aorta Radiology, *12* 58, 1929

Ratschow, M, and Arendt, J Zur Diagnose der Isthmusstenose der Aorta Fortschr a d Geb. d Rontgenstrahlen, *49* 347, 1934

Rautmann, H Untersuchungen uber die Norm Ihre Bedeutung und Bestimmung. Veroff Kriegs-u Konstit path, *2* 6, 1921

Rautmann, H Zur Frage der Herzgrosse bei Sportleuten. Klin Wchnschr, *6* 23, 1927

Rautmann, H Wirkungen des sportlichen Laufes Ztschr f klin med, *98* 58, 1924

Rautmann, H Die Wirkung sportlicher Tatigkeit auf die Kreislauf- und Atmungsorgane sowie den Stoffwechsel Dtsch med Wchnschr., *59* 1238, 1278, 1932

Rautmann, H Ueber das Sportherz Med Klin, *31* 1018, 1935

Rautmann, H, and Heiss, F Zur Kenntnis der korrelativen Variabilitat der orthodiagraphischen Herzgrosse Ztschr Konstit forsch, *13* 567, 1928

Rauch, H. Ueber die Darstellbarkeit und Form der Aortensklerose sowie der Verkalkungen der Herzklappen im Rontgenbild Ein Fall von rontgenologisch dargestellten verkalkten Herzthromben Munchen, Diss, 1938

Ravina, A L'exploration radiologique des vaisseaux pulmonaires par l'injection de substance de contraste Progrès méd, *42* 1701, 1934

Regelsberger, H Ein typischer Fall von Herzaneurysma Rontgenpraxis, *6* 806, 1934

Rehn, L Zur Chirurgie des Herzens und des Herzbeutels Arch f klin Chir, *83* 723, 1907

Reich, L Das Rontgenbild und die orthodiagraphische Messung der Aorta im 2 schragen Durchmesser Fortschr a d Geb d Rontgenstrahlen, *34* 322, 472, 1926

Reifenstein, E C, and Allen, E G Aneurysm of the thoracic aorta Report of a case presenting some unusual features Am Heart J, *6* 274, 1931

Reinberg, S A, and Lindenbraten, D S Anatomisch-rontgenologische Studien zur Frage von der Pericarditis exsudativa im fruhen Kindesalter. Fortschr a d Geb d Rontgenstrahlen, *49* 598, 1934

Reindell, H. Diagnostik der Kreislauffruhschaden, Stuttgart, Enke, 1949.

Reindell, H Elektrokardiographische und kymographische Untersuchungen am Sportler nach Belastungen Verh dtsch Ges Kreislaufforsch, 1937, p 275

Reindell, H Kymographische und elektrokardiographische Befunde am Sportherzen I Mitt Dtsch Arch f klin Med, *181* 485, 1938, II Mitt, ebenda, *182* 506, 1938.

Reindell, H Die Herzbeurteilung beim Sportsmann, usw. Dtsch med. Wchnschr, *65* 1369, 1939

Reindell, H Herz und Sport Fortschr. a d Geb d Rontgenstrahlen, *60* 35, 1939

Reindell, H Grosse, Form und Bewegungsbild des Sportherzens Arch Kreislaufforsch, *7* 117, 1940

Reindell, H Kymographische Beobachtungen uber der erhohte Restblutmenge beim Gesunde Verh. dtsch Ges Kreislaufforsch, *13*. 144, 1941.

Reinhart, P Rontgenbefunde bei Beri-Beri Fortschr a d. Geb d Rontgenstrahlen, *24* 104, 1916–7.

Reitan, H Beitrag zur Rontgendiagnose der Perikarddivertikel und der abgesackten Perikardexsudate. Fortschr a d Geb d Rontgenstrahlen, *58* 195, 1938.

Renck, G Ueber das Renalisaneurysma, besonders vom rontgenologischen Gesichtspunkt. Acta radiol, 7.309, 1926

Reuterwall, O Zur Frage der Arterienelastizitat. Arch f path Anat, *239* 363, 1922.

Reynolds, R. J Cineradiography Brit J Radiol, 7 415, 1934

Rieder, H Das "Panzerherz" Fortschr a d Geb. d Röntgenstrahlen, *20* 50, 1913.

Rieder, W. Herzschadigung infolge arteriovenosen Aneurysmas Arch f klin Chir, *139* 597, 1926

v Ritook, Z Ueber Hypoplasie des Arteriensystems Ztschr. f klin Med, *61*.171, 1907.

Robb, G P., and Steinberg, I. Visualization of the chambers of the heart Am J Roentgenol., *41* 1, 1939.

Robb, G P, and Steinberg, I.· Visualization of the chambers of the heart, the pulmonary circulation and the great blood vessels in heart disease, preliminary observations Am J Roentgenol., *42*.14, 1939

Roessle, R., and Roulet, F. Mass und Zahl in der Pathologie Berlin, Springer, 1932.

Rohrer, F.: Volumbestimmung von Korperhohlen und Organen auf orthodiagraphischen Wege. Fortschr a d. Geb d Rontgenstrahlen, *24* 285, 1916–7

v. Rokitansky, C. Die Defekte der Scheidewande des Herzens. Wien, Braunmuller, 1875

Rosenhagen, H Ueber einige Beziehungen zwischen histologischen Veranderungen und Rontgenbild bei der chronischen Stauungslunge Fortschr a d Geb d Rontgenstrahlen, *38* 353, 1928.

Rosler, H Zur röntgenologischen Beurteilung des Herzgefassbildes bei Thoraxdeformitaten (Kypho-Skoliosis, reine Kyphose, Trichterbrust) Dtsch Arch f klin Med, *164* 365, 1928

Rosler, H. Beitrage zur Lehre von angeborenen Herzfehlern I–IV Mitteilung Wien Arch inn Med, *15* 487, 1928

Rösler, H Das Rontgenbild des Herzens beim Hyperthyreoidismus Wien Arch f inn Med, *15* 125, 1928

Rosler, H · Rechtseitige mitgeteilte Hiluspulsation bei aneurysmatischer Erweiterung des linken Vorhofs Fortschr a d Geb d Rontgenstrahlen, *40* 1017, 1929

Rösler, H Ueber Herzvergrosserung bei angeborenen arteriovenoser Kommunikation Klin Wchnschr, *8* 1621, 1929

Rosler, H. Beitrage zur Lehre von den angeborenen Herzfehlern Wien Arch. f inn. Med, *19* 505, 1930

Rosler, H. Die Grenzen des Normalen und Pathologischen im Rontgenbild des Herzens und der grossen Gefasse Klin Wchnschr, *9* 607, 1930

Rösler, H Beitrage zur Lehre von den angeborenen Herzfehlern VI Ueber die angeborene isolierte Rechtslage des Herzens Wien Arch f inn Med, *19* 605, 1930.

Roesler, H. The relation of the shape of the heart to the shape of the chest Am J Roentgenol., *32* 464, 1934

Roesler, H Interatrial septal defect Arch Int Med., *54* 339, 1937

Roesler, H Exercise and the heart J Health and Physical Education, 7 141, 1936

Roesler, H Clinical Roentgenology of the Cardiovascular System Springfield, Thomas, 1946

Roesler, H, Gifford, U G, and Betts, W Dissecting aneurysm of the aorta, correctly diagnosed. Am Heart J, *13* 426, 1937

Rosler, H, and Weiss, K Ueber die Veranderung des Oesophagusverlaufes durch den vergrosserten linken Vorhof Fortschr a d Geb d Rontgenstrahlen, *33* 417, 1925

Roesler, H, and White, P D Unusual variations of the roentgen shadow of the elongated thoracic aorta Am Heart J, *6* 768, 1931

Rossi, E, and Prader, A Die Angiokardiographie bei angeborenen Herzfehlern Schweiz med Wchnschr, *56* 1124, 1948.

Rossi, E, and Rohner, M Un cas de tetralogie de Fallot avec arc aortique droit et atrésie du tronc brachiocephalique veineux gauche Helv paed acta, *3* 124, 1948

Rothberger, J Herztonus Handbuch der Physiologie, Bd 7/1 Berlin, Springer, 1927–32.

Routier, D, and Heim de Balsac, R Étude sur le deroulement aortique. Bull. Soc Radiol. méd. France, *21* 35, 1933

Routier, D, and Heim de Balsac, R Nature, place et interpretation de la sémiologie radiologique Presse méd, *41* 1642, 1934

Routier, D., Heim de Balsac, R, and Gerbeaux, J Étude anatomoradiologique des *artères coronaires* par le radiographie après opacification Arch d mal du coeur, *31* 441, 1938

Ruffin, M, Castleman, B, and White, P D Arteriosclerotic aneurysms and senile ectasia of the thoracic aorta Am Heart J, 22 458, 1941

Ruhl, A Ueber Herzinsuffizienz durch Histamin Arch exper path, *174* 755, 1934

Rummert, O Ueber Perikardverkalkungen Fortschr a d Geb d. Rontgenstrahlen, *55* 241, 1937.

Rumpf, T Rontgenuntersuchungen bei abnorm beweglichem Herzen (Wanderherz) Dtsch Arch f klin Med, *129* 118, 1919

Rusca, F Experimentelle Untersuchungen über die traumatische Druckwirkung der Explosionen Zentralbl f Chir, *139* 315, 1914.

Russek, H J, and Zohman, B L The syndrome of Bernheim as a clinical entity Circulation, *1* 759, 1950.

S

Sabat, B Zur Geschichte der Rontgenkymographie und Ausarbeitung der Modifaktion der Methode. Fortsche a d Geb d Rontgenstrahlen, *50* 309, 1934
Sachs, H Ein Aneurysma der Aorta descendens mit pulsierender Verwolbung unterhalb der linken Skapula Fortschr. a d Geb d Rontgenstrahlen, *31* 285, 1923–4
Sahatschieff, A Beitrag zur Rontgenuntersuchung des Herzens. Fortschr. a d Geb d Rontgenstrahlen, *33* 683, 1925
Salinger, H Die Knochenbildungen in der Lunge mit besonderer Berucksichtigung der tuberosen Form Fortschr a d Geb d Rontgenstrahlen, *46* 270, 1932
Salotti, R Ricerche radiologiche sulla influenza immediata del'atto operativo e della narcosi sul cuore Radiol med, *15* 388, 1928
Salzer, G Kontraststellung der Verengerung der oberen Hohlvene Ges. d Aertze Wien, 1948
Sante, L. R, and Wyatt, J P. Roentgenological and pathological observations in antigenic pneumonitis Am J Roentgenol, *66* 527, 1951.
Satke, O Ueber den Hydrothorax bei kardialer Insuffizienz Ztschr. f klin Med *113* 202, 1930
Sauerbruch, F, Chaoul, H, and Adams, A Anatomisch-klinischer und rontgenologischer Beitrag zur "Hiatushernie" Dtsch. med Wchnschr., *58* 1391, 1932
Saul, W. Verdichtungen im Herzen Fortschr a d Geb d Rontgenstrahlen, *46* 450, 1932.
Saupe, E Pneumoperikard mit linksseitigem Pneumothorax. Fortschr. a d Geb d Rontgenstrahlen, *27* 488, 1919–21.
Savini, E Ueber die radioskopische Diagnose der Pulmonalarteriensklerose, Beschreibung eines neuen Rontgenphamonens bei derselben Zentralbl. f Herz u Gefasskrkh, *7* 148, 169, 1915
Schaede, A Trikuspidalatresie bei einem 21-jahrigen Manne Ztschr f. Kreislaufforsch., *41* 132, 1952
Schaefer, G Das Herz bei Trichterbrust Ztschr f Kreislaufforsch, *26* 689, 1934
Schatski, R Plastische grossen und lagewahre Darstellung des Herzens Fortschr a d Geb d Rontgenstrahlen, *37* 889, 1928
Schatski, R Das gestielte Aortenaneurysma Beitrag zur Differentialdiagnose von Mediastinaltumor und Aneurysma Fortschr a d. Geb d Rontgenstrahlen, *44* 348, 1931
Schatski, R., and Hallermann, W Ueber die Isthmusstenose der Aorta Fortschr a d Geb d Rontgenstrahlen, *42* 324, 1930
Scheef, S Ueber die Ruptur eines mykotischen Aneurysma des Ductus Botalli und die rontgenologische Darstellung des erweiterten Ductus beim Saugling Arch f Kinderh, *117* 234, 1939
Schellong, F Storung der Kreislaufregulation, ein neues Symptom bei Insuffizienz des Hypophysenvorderlappens Klin Wehnschr, *10* 100, 1931
Schellong, F Weiteres uber das Symptom der Blutdrucksenkung nach Korperarbeit. Klin. Wchnschr., *11* 53, 1932
Schellong, F, and Heinemeier Ueber die Kreislaufregulation in aufrechter Korperstellung und ihre Storungen I and II Mitteilung. Ztschr f exper. Med, *89* 49, 1933
Scherf, D, and Boyd, L J Cardiovascular Diseases Philadelphia, Lippincott, 1947
Scherf, D Ueber die relative Insuffizienz der Pulmonalklappen Klin Wchnschr, *9* 868, 1930.
Scherf, D Klinik und Therapie der Herzkrankheiten Wien, Springer, 1936
Scherf, D, and Schonbrunner Ueber Herzbefunde bei Lungenembolien Ztschr f klin Med, *128* 455, 1935
[illegible] von echtem Herzalternans beim [illegible] 929
[illegible] grosse durch Atropin, Adrenalin [illegible]
[illegible] klin Med, *89* 604, 1907.
[illegible] ch Arch. f klin Med, *92* 392, 1908.
Schiff, E. Konstitutionelle Schwache des Zirkulationsapparats im Kindesalter. Jahrb d Kinderh, *91* 217, 1920

Schilling, C.: Die Anwendung der Flachenkymographie in der Diagnostik der Herzerkrankungen Fortschr a d. Geb d Rontgenstrahlen, 47 241, 1933

Schirmer, O. Ueber Pericarddivertikel Zentralbl d. Path, 34 143, 1923-4.

Schlapper, K. Ueber Form und Formveranderung des Herzens bei Lungentuberkulose unter besonderer Berucksichtigung des Teleorontgenogramms. München med Wchnschr, 78 1290, 1931

Schlesinger, H Syphilis und innere Medizin. III Teil Wien, Springer, 1928.

Schlesinger, H Zur Symptomatologie und Fruhdiagnose der Aortenlues. Wien Arch f inn Med., 26,181, 341, 1935.

Schlomka, G: Experimentelle Untersuchungen uber den Einfluss stumpfer Brustkorbtraumen auf des Herz V Das besondere Verhalten sensibilisierter Tiere Ztschr f exper Med, 92 552, 1934

Schlomka, G, and Daum, H Ueber die Spontanschwankungen der Herzgrosse beim Gesunden. Fortschr a.d. Geb d Rontgenstrahlen, 55 558, 1937

Schlomka, G, and Schmitz, M Experimentelle Untersuchungen uber den Einfluss stumpfer Brustwandtraumen auf das Herz IV Die akute traumatische Herzdilatation Ztschr f exper. Med., 90 301, 1933

Schludermann, H Ueber kongenitale und erworbene periphere Aneurysmen der Arteria pulmonalis Fortschr a d Geb d Rontgenstrahlen, 76 8, 1952

Schmidt, E A The heart in pulmonary tuberculosis Radiology, 21 167, 1933

Schmidt, W Experimentelle Untersuchungen zur Kernschattenfrage bei Pericarditis exsudativa Fortschr a d Geb d Rontgenstrahlen, 55 351, 1937

Schmieden, V, and Fischer, H Die Herzbeutelentzundung und ihre Folgezustande Ergebn Chir u Orthop., 19 98, 1926

Schmitt, H Mittellappen der linken Lunge und seltene Form des Lobus venae azygos Rontgenpraxis, 10 568, 1938

Schneider, J Infantile Herzhypertrophie (Beitrag zur Frage Glykogenspeicherungskrankheit) Helvet paediat acta, 1 304, 1946

Scholz, T Radiographic demonstration of myocardial calcification during life J Radiol, 5 131, 1924

Scholz, T Rontgenologische Darstellung von Herzthromben Fortschr a d Geb d Rontgenstrahlen, 32 416, 1924

Schoppler, H Ueber ein grosszelliges Rundzellensarkom des Mediastinums Frankf Ztschr f Path 22 305, 1919

Schultze, F Pericarditis exsudativa im Rontgenbilde Dtsch med Wchnschr, 7 863, 1921.

Schultz, M, and Klinge, F Das Gewebsbild des fieberhaften Rheumatismus Aortitis rheumatica und Arteriosklerose Arch f path Anat, 288 717, 1933

Schur, M Probleme der adhasiven Perikarditis Ergebn. d. inn Med, 47 548, 1934

Schutze, J 2 Falle von Granatsplitter im Herzens Dtsch med Wchnschr, 42 515, 1916

Schwartz, S P The radiographic signs of pulmonic insufficiency. Am. Heart J, 2 407, 1926-7

Schwartz, S P Pericardial effusion following acute coronary vessel closure Am Heart J, 10 253, 1934

Schwartz, S P, and Shelling, D Acquired rheumatic pulmonic stenosis and insufficiency. Am. Heart J, 6 568, 1930-1

Schwarz, G Rontgenoskopische Beobachtungen von Eigenpulsationen der Hilusschatten und ihrer Verzweigungen. Wien klin Wchnschr, 23 892, 1910.

Schwarz, G Ueber einen typischen Rontgenbefund am Herzen Fettleibiger. Wien klin Wchnschr, 23 1850, 1910

Schwarz, G Die Rontgenuntersuchung des Herzens und der grossen Gefasse Wien, Deuticke, 1911

Schwarz, G. Demonstration einer ausgedehnten Kalkplatte im Herzbeutel, mittels Rontgenuntersuchung nachgewiesen, unter dem Bilde einer Lebercirrhose verlaufend Wien klin Wchnschr, 24 1541, 1911.

Schwedel J B, and Gross, H

Schwedel, J B, and Gutman,
with roentgen and post m

Scott, R. W Aortic aneurysm rupturing into the pulmonary artery J.A.M.A., *82* 1417, 1924

Secher, L Experimentelle Untersuchungen uber die Grosse des Herzens nach einem Aufhoren des Trainierens Ztschr f exper. Med , *32* 290, 1923

Seckel, H. Kreislaufsystem und zirkulierende Blutmenge bei kranken Kindern III. Mitteilung· Die orthostatische Vasoneurose der alteren Kinder Jahrb f Kinderh , *137* 51, 1932

Seidler, E Ueber Perikarddivertikel *Wien klin Wchnschr*, *34* 592, 1921

Seuderling, Y, and Kumlin, T A rare combination of anomalies of the aorta (Two cases of arcus aortae dexter circumflexus combined with elongation and dilatation of the descending aorta) Brit J Rad , *25* 165, 1952

Shookhoff, C , and Douglas, A H A case of acute coronary occlusion with roentgenographic evidence of the early development of an aneurysm of the left ventricle Am Heart J , *7* 95, 1931

Simmonds, M Ueber den Nachweis von Verkalkungen im Herzen durch das Rontgenverfahrens Fortschr a d Geb d Rontgenstrahlen, *12* 371, 1908

Simonsen, M Rundschatten in der Lunge Rontgenpraxis, *8* 476, 1936

Smith, H L , and Horton, B T. Arteriovenous fistula of the lung associated with polycythemia vera Am Heart J , *18* 589, 1939

Snellen H A , and Nauta, J H Zur Rontgendiagnostik der Koronarverkalkungen Fortschr a d Geb d Rontgenstrahlen, *56* 277, 1937

Snelling, C. E , and Erb, J. H Double aortic arch Arch Dis Childh , *8* 401, 1933

Sosman, M C , and Wosika, P H Calcification in aortic and mitral valves Am. J. Roentgenol , *30* 328, 1933

Sosman, M. C , and Wosika, P H The position of the heart valves and their relation to the anterior chest wall in living subjects with abnormal hearts Am Heart J , *10* 156, 1934

Sotter, A Herzgrosse und Asthmakonstitution *Fortschr a d Geb d Rontgenstrahlen, 58 89, 1938.*

Soulié, P , Nouaille, J , Schweisguth, O , and Touche, M Le "truncus aorticus" (diagnostic clinique et radiologique) Bull mem Soc méd hôp Paris, *66* 919, 1950.

Sparks, J V, and Evans, C Radiography of calcification in cardiac valves during life Brit. Med J , *1* 1028, 1934

Spencer, H , and Dworken, H J Congenital aortic septal defect with communication between aorta and pulmonary artery. Circulation, *2* 880, 1950

Spencer, J , and Dresser, R Right-sided aorta Am J Roentgenol , *36* 13, 1936

Spiro, H Determination of the quality of the heart muscle by fluoroscopy Radiology, *1* 229, 1923

Spitzer, A Ueber den Bauplan des normalen und missbildeten Herzens. Arch f. path Anat , *243* 81, 1923

Spitzer, H Fusiformes Aneurysmas der Arteria pulmonalis bei Dreiklappenfehler Fortschr a d. Geb d Rontgenstrahlen, *36* 1236, 1927.

Spitzer, H Die unregelmassige Herztatigkeit am Rontgenschirm Fortschr. a d Geb. d *Röntgenstrahlen*, *39* 126, 1929

Spycher, C *Rontgenographische Untersuchungen der menschlichen Herzmasse* bei stark verminderten Luftdruck. Arb physiol , *4* 390, 1931

Stadler, E Ueber Isthmusstenose der Aorta bei syphilitischer Aortenerkrankung. Zentralbl f Herzkrkh , *13* 357, 1921

Starck, H Zur Pathologie des Panzerherzens. Med Klin , *24* 1666, 1928

Stauffer, H M , and Rigler, L Dilatation and pulsation of the left subclavian artery in the roentgen ray diagnosis of coarctation of the aorta Circulation, *1* 294, 1950

Steel, D The roentgenological diagnosis of cardiac aneurysms J A M A , *102* 432, 1936

Steffens, W Herzsteckschusse Leipzig, Thieme, 1936

Stehr, L., and Grottker, G Freie und abgekapselte Herzbeutelergusse Med Welt , *12* 1413, 1938

Steinberg, M F , and Weisner Beobachtungen von Morbus coeruleus. Ztschr f klin Med , *129*.231, 1935.

[illegible] Nonsyphilitic aneurysm of the aorta in [illegible]

[illegible] phy in congenital heart diseases III Patent Ductus arteriosus Am J Roentgenol , [illegible], 1943.

Steinberg, M. F., Grishman, A., and Sussman, M L Non-luetic aortic aneurysm, angiocardiographic diagnosis J Mt Sinai Hosp., *10* 733, 1944

Steiner, G. Ueber das "Aneurysma der Arteria pulmonalis" Röntgenpraxis, *7* 168, 1935.

Steiner, O Beziehungen zwischen Kropf und Herz. Ihr Verhalten nach der Strumektomie. Mitt. Grenzgeb Med u Chir., *35* 39, 1922

Stender, H S Die Rontgensymptome der Arteritis pulmonalis und ihrer Folgezustande Fortschr a d Geb d Rontgenstrahlen, *76* 316, 1952

Stenstrom, N G, and Westermark, N A study of the activity of the human heart simultaneously recorded by x-rays and electrocardiogram Acta radiol, *5* 408, 1926

Steuer, L G, and Higley, C S Primary sarcoma of pericardium J A M A, *105* 1110, 1935

Stewart, H J, and Cohn, A F Studies on the effect of the action of digitalis on the output of blood from the heart J Clin Investigation, *11* 897, 917, 1932

Stolte, K. Die Erkrankungen des Herzens usw In Pfaunder-Schlossmann Handbuch der Kinderheilkunde, Bd 3, Berlin Vogl, 1931

Stoerck, O. Zur Topographie des Mediastinums bei normaler und pathologischer Herzform Ztschr f klin Med, *69* 32, 1910

Stormer, A. Verschwinden klinisch bestandener Zeichen eines kombinierten Mitralvitiums Munchen med Wchnschr, *76* 1328, 1929

Strisower, R Ueber bedeutende Blutdrucksenkung nach Arbeit und bei Aenderung der Korperlage bei Tabes dorsalis Ztschr f klin Med, *117* 384, 1931

Strothmann Ueber einen Fall von isolierter, komplizierter Dextrokardie mit corrigierter Transposition der grossen Gefasse Dtsch Arch f klin Med, *163* 76, 1929

Stumpf, P Die Gestaltanderung des schlagenden Herzens im Rontgenbild Fortschr a d Geb d Rontgenstrahlen, *38* 1055, 1928

Stumpf, P Die Erscheinungsformen der Herzmuskelerkrankungen Flachenkymogram Fortbildg lehrg Nauheim, 1934

Stumpf, P Ruckblick zur Kymographie des Herzens Fortschr a d Geb d Rontgenstrahlen, *68* 1055, 1943

Stumpf, P, Weber, H H, and Weltz, G A Rontgenkymographische Bewegungslehre innerer Organe, Leipzig, Thieme, 1936

Sulger, E Experimentelle Untersuchungen uber den Einfluss der Trachealstenose auf Herz und Kreislauf (Ein Beitrag zur Frage Gibt es ein mechanischer Kropfherz und wie entsteht es?) Dtsch Ztschr f Chir *201* 21, 1927.

Sunderberg, C G The roentgen kymogram of the normal heart. Acta radiol, *14* 558, 1933

Sussman, M L, and Brahms, S A Interpretation of normal cardiovascular angiograms with discussion of common errors Am J Roentgenol, *66* 29, 1951

Sussman, M L, Dacks, S, and Master, A M The roentgenkymogram in myocardial infarction I The abnormalities in left ventricular contraction Am Heart J, *19* 453, 1940

Sussman, M L, and Grishman, A A discussion of angiocardiography Advances in Internal Medicine, *2* 102, 1947

Suter, F Ueber das Verhalten des Aortenumfanges unter physiologischen und pathologischen Bedingungen Arch f exper Path, *39* 289, 1897

Svanberg, T Roentgenographical pulmonary changes in periarteritis nodosa Acta radiol, *26* 307, 1945

Sylla, A Lungenstauung und Stauungslunge Ergebn inn Med u Kinderh, *49* 122, 1935

T

Taipale, L Ueber einen neuen rontgenologischen Index der Herzgrosse Duodecim, *43* 32, 1927.

Talley, J E, and Fowler, K. Tetralogy of Fallot (Eisenmenger type) with hypoplasia of the dextroposed aorta Am J. M. Sc., *191* 618, 1936

Tallquist, T W Ist das hypoplastische Herz einer kompensatorischen Hypertrophie fahig? Kongr dtsch Ges inn Med, 1922, 219

Tamiya, C. Ueber ein neues Prinzip für Grossenbestimmung des Herzens seine praktische Anwendung Fortschr a d Geb. d Rontgenstrahlen, *41* 62, 1930.

Taussig, H B The clinical and pathological findings in congenital malformations of the heart due

to defective development of the right ventricle associated with tricuspid atresia or hypoplasia Bull. Johns Hopkins Hosp, *59* 435, 1936

Taussig, H. B Congenital Malformations of the Heart. New York, The Commonwealth Fund, 1947

Taussig, H. B Clinical and pathological findings in cases of truncus arteriosus in infancy Am J Med, *2* 26, 1947

Taussig, H B, and Bing, R J *Complete transposition of the aorta and levoposition of the pulmonary artery* Am Heart J, *37* 551, 1949

Taylor, B E, and DuShane, J. W Patent ductus arteriosus associated with pulmonary stenosis. Proc Staff Meetings Mayo Clin, *25* 60, 1950

Temple, H L, Steinberg, I., and Dotter, C T Angiocardiography utilizing photoroentgen apparatus with rapid film changer Am J Roentgenol, *60* 646, 1948

Teschendorf, W · Lehrbuch der rontgenologischen Differentialdiagnostik 3 Aufl, Stuttgart, *Thieme, 1952*

Teschendorf, W Lehrbuch der rontgenologischem Differentialdiagnose der Erkrankungen der Brustorgane, Leipzig, Thieme, 1929

Teschendorf, W. Rontgenologische Herzfunktionsprufungen Fortschr a.d Geb d Rontgenstrahlen, *60* 17, 1929.

v Teubern, K Orthodiagraphische Messungen des Herzens und des Aortenbogens bei Herzgesunden. Fortschr a d Geb d Rontgenstrahlen, *24* 549, 1916–7.

Thoma, R [illegible] 1888, *112* 259, 383, 1888, C [illegible]

Thums, K [illegible] 4, 1933

Thurn, P Rontgenkymographische Befunde bei kongenitalen Herzfehlern Fortschr. a d Geb. d Rontgenstrahlen, *74* 151, 1951

Thurnher, B. Die angeborenen Anomalien der Aorta thoracica im Rontgenbild Wien Ztschr f inn. Med, *32* 132, 1951

Thurnher, B., and Weissel, W Links randbildende Aorta descendens bei Morbus coeruleus Cardiologia, *18* 45, 1951

Tillich, A. Perforation eines *mykotischen Aneurysmas* bei Isthmusstenose Rontgenpraxis, *13* 143, 1941

Todtenhaupt Kongenitaler Herzfehler (Pulmonalstenose und Ventrikelseptaldefekt, Transpositio aortae) und Erythrozytose Dtsch Arch f klin Med, *154* 218, 1937

Tompkins, R D Aneurysm of left aortic sinus (Valsalva) with rupture into right ventricle M *Bull Vet Admin, 18 173, 1941.*

Traugott, K Zur Diagnose der Herzbeutelergusse Munchen med Wchnschr, *67* 1010, 1920

Traum, E Beitrag zur Bedeutung des Aneurysmas des Ductus arteriosus Botalli Chirurg, *2* 618, 1930

Trendelenburg Schussverletzung des Herzens mit Einheilung der Kugel Verh dtsch Ges Chir., 31 Kongress, 1902, *26*

Tschilow, K, and Christoff, E. Die Wirkung des Aderlasses auf die Herzgrösse Ztschr f exper *Med, 89 173, 1933.*

Tullio, P, and Businco, O.· Die Veranderungen des Herzumfanges in der akuten Asphyxie. Fortschr a d. Geb d Rontgenstrahlen, *29* 291, 1929.

Tung, C. L, Bien, W. N, and Ch'u, Y C The heart in severe anemia Chin M. J, *52* 479, *1937.*

Tung, C L., Hsieh, C. K, Bien, C W, and Dieuaide, F R. The heart of ricksha pullers. Am Heart J, *10* 77, *1934*

U

[illegible] der Herzens bei Erkrankungen der Lungen [illegible] 1935

[illegible] nerkrankungen *Fortschr.* [illegible]

[illegible] all von Pulmonalstenose

Uhlenbruck, W., Untersuchungen [illegible] Ztschr. f. Kreislaufforsch, *19* 601, 1927

Urbach, J Die Verletzungen des Herzens durch stumpfe Gewalt Beitr z gerichtl Med, *4* 104, 1922.

Usomoto, S. Ein Beitrag uber das Rontgenbild der Pulmonalstenose, inbesondere uber die Vorwolbung des linken zweiten Bogens Dtsch Arch f klin. Med, *147* 159, 1925

V

Van Liere, E J The effecto of anoxemia on the size of the heart as studied by x-ray. Am. J Physiol, *82* 727, 1927

Vaquez, H, and Bordet, E Radiologie du Coeur et des Vaisseaux de la Base. Paris, Bailliere, 1928.

Veith, A Ueber orthodiagraphische Herzuntersuchungen bei Kindern im schulpflichtigen Alter Jahrb Kinderh, *68* 203, 1908

Velde, G Ein eigentumlicher Schattenreifen in der rechten Lungenspitze Fortschr. a d Geb d Rontgenstrahlen, *36* 315, 1927.

Vierordt, H Stenosen und Obliteration der Aorta an der Einmundung des Ductus arteriosus (Persistenz der Isthmus aortae) Nothnagel's Handbuch Bd 15, 1898

Vierordt, H Die angeborenen Herzkrankheiten Nothnagel's Handbuch Bd 15, 1901

Vogl, A Ein Fall von luischem Aneurysma der Arteria pulmonalis Med Klin, *27* 1352, 1931.

Vogt, A Die Isthmusstenose der Aorta Fortschr a d Geb d Rontgenstrahlen, *74* 159, 1951.

Vogt, W Herzaneurysma Rontgenpraxis, *9* 219, 1937

W

Wahl, H R, and Gard, R L Aneurysm of the pulmonary artery Surg, Gynec and Obst, *52* 1129, 1931

Walldén, L Ueber Rontgensymptome des exsudativer Perikarditis Uppsala Lak Forh, *48* 687, 1942

Warfield, C H Roentgen diagnosis of aneurysms of the innominate artery. Am J. Roentgenol, *33* 350, 1935

Weber, A Beobachtungen am traumatischen Aneurysma arteriovenosum Munchen med Wehnschr, *64* 409, 1917

Weber, A Eine Methode zur Darstellung von Herzmomentaufnahmen in verschiedenen Phasen der Herzrevolution 27 Kong Inn Med, Wiesbaden, 1910, 673

Wechsler, Z Die "pleuritischen" Mediastinalstreifen im Kindesalter in ihrer klinischen Bedeutung Fortschr a d Geb d Rontgenstrahlen, *44* 342, 1930

Wedler, H W Multiple mykotische Aneurysmen der Art pulmonalis Fortschr. a d Geb d Rontgenstrahlen, *68* 141, 1944

Wegemer, E Das Pneumoperikard als Komplikation im Gefolge der Pneumothoraxbehandlung Beitr z Klin Tuberkl, *92* 437, 1938

Weil, A Panzerherz und Picksche Leberzirrhose. Fortschr a d Geb d Rontgenstrahlen, *23* 195, 1915–6

Weinberger, M Atlas der Radiographie der Brustorgane Wien, Engel, 1901

Weinberger, M Weitere Beitrage zur Radiographie der Brustorgane Med Klin, *4* 584, 1908

Weinberger, M Zur Klinik der angeborenen isolierten Dextrokardie und Dextroversio cordis Zentralbl f Kerzkrkh, *11* 121, 137, 1919

Weinbren, M A Manual of Tomography. Springfield, Charles C Thomas, 1948

Weiser Fall von Panzerherz Med Klin, *22* 1093, 1926.

Weiss, E Calcified plaque of aorta at entrance of patent ductus arteriosus point in diagnosis Am Heart J, *7* 114, 1931

Weiss, K Ueber teleradiologische Vergleichsaufnahmen Wien med Wchnschr, *70* 550, 595, 692, 1920

Weiss, S, Kinney, T D, and Maher, M M Dissecting aneurysm of the aorta with experimental atherosclerosis Am J M. Sc, *200* 192, 1940

Weltz, G A Die Bewegungen des sagittalen Herz- und Aortenbildes bei der Atmung Fortschr. a d Geb d Rontgenstrahlen, *50* 153, 1934

Weltz, G A Die pulsatorischen Bewegungen der Brustaorta Fortschr a d Geb d Rontgenstrahlen, *51* 152, 1935

Wenckebach, K F Ueber Herzkrankungen bei Kriegteilnehmern Verh dtsch Ges inn Med, 1916, 50

Wenckebach, K F Ueber pathologische Beziehungen zwischen Atmung und Kreislauf beim Menschen Sammlg klin. Vortr, 1907/1909, Nr 465/466

Wenckebach, K. F Beobachtungen bei exsudativer und adhasiver Perikarditis Ztschr f klin. Med. *71* 402, 1910

Wenckebach, K F · Ueber pathologische Atmungs- und Thoraxformen Wien Arch f. inn Med, *1* 1, 1920

Wenckebach, K F Das Beriberi-Herz Berlin-Wien, Springer, 1934

Werkenthin, M The roentgenological aspect of lung edema Am J Roentgenol, *41* 183, 1939

Westermark, N Studien uber die Bewegungen des Herzens Acta radiol, *17* 235, 1936

Westermark, N *On the roentgenological diagnosis of lung embolism Acta radiol, 19 357, 1938*

Weymuller, C A., Bell, A L L, and Krahulick, L Roentgenographic changes in the thorax of normal newborn babies Am J Dis Child, *35* 837, 1928

White, P. D Heart Disease, New York, Macmillan, 1952

White, P D, and Boyes, J H Subacute bacterial (*Streptococcus viridans*) *endocarditis* and endarteritis involving the tricuspid valve and the pulmonary artery in a unique case of the tetralogy of Fallot complicated by congenital pulmonary regurgitation Am. Heart J, *7* 802, 1931

White, P D, and Camp, P D A comparison of orthodiagraphic and teleroentgenographic measurements of the heart and thorax. Ann Int Med, *6* 469, 1932.

White, P. D, and Camp, P. D : Pericardial effusion A clinical study. Am J M Sc, *184* 328, 1932.

Wierig Beitrage zum Kapitel der Lungenzeichnung im Rontgenbild Fortschr a d Geb d Rontgenstrahlen, *35* 704, 1927

Wiese, O Weitere Beitrage zur Kenntnis "benigner" intrathorakaler Tumoren Beitr z Klin Tubkl, *78* 421, 1931

Wiese, O Ueber maximale Herzverlagurgen beim Kinde als Folge langsam verlaufender Lungetuberkulose mit besonderer Berucksichtigung der Dextrokardie (Dextroversio cordis) Beitr z Klin Tubkl, *68* 143, 1928.

Wiberg, G A case of aneurysm of the heart Acta radiol, *12* 562, 1931

Wilkens, G D Ein Fall von multiplen Pulmonalisaneurysms Beitr z Klin Tubkl, *38* 1, 1918

Williams, R G, and Steinberg, I. Value of angiocardiography in establishing the diagnosis of *pericarditis with effusion Am J Roentgenol, 61 41, 1949*

Williamson, C S The effects of exercise on the normal and pathological heart, based upon the study of one hundred cases Am J M Sc, *149* 492, 1915

Williamson, C S Pericarditis with effusion An experimental study. Arch Int. Med, *25* 206, 1920.

Wilson, C Physiological dilatation of the heart Brit. M J, *1* 189, 1930

Wittenborg, M H, Neuhauser, E B, and Sprunt, W H. Roentgenographic findings in congenital tricuspid atresia with hypoplasia of the right ventricle. Am J Roentgenol, *66* 712, 1961

Wolke, K Two cases of coarctation (stenosis of the isthmus) of the aorta Acta radiol, *18* 319, 1937

Wood, F C, Pendergrass, E P, and Ostrum, H W Dissecting aneurysm of the aorta with special reference to the roentgenographic features Am J Roentgenol, *28* 437, 1932.

Wosika, P. H, and Sosman, M. C The roentgen demonstration of calcified coronary arteries in *living subjects J A M A., 102 591, 1934*

Wright, A. D Calcified cyst of the pericardium Brit J. Surg, *23* 612, 1936

Y

Yater, W. M. Cyst of the pericardium. Am Heart J, *6* 710, 1931.

Z

Zadek, E. Zur Diagnose der Coronarsklerose Klin Wchnschr, *11* 1255, 1932.

Zadek, L. Zur Diagnose der Pulmonalinsuffizienz und stenose Fortschr. a d Geb. d Rontgenstrahlen, *23* 326, 1915–6

Zdansky, E Mediastinalwandern bei Skoliose der Wirbelsaule Fortschr a d Geb d Rontgenstrahlen, 37 172, 1927
Zdansky, E Beiträge zur Kenntnis der kardialen Lungenstauung auf Grund rontgenologischer, klinischer, und anatomische Untersuchungen Wien Arch f inn Med, 18 461, 1929
Zdansky, E Ueber das Rontgenbild der kardialen Lungenstauung Fortschr a d Geb d Rontgenstrahlen, 42 746, 1930
Zdansky, E Zur Diagnose der Concretio und Accretio cordis Fortschr a d Geb d Röntgenstrahlen, 44 48, 1931
Zdansky, E Aenderungen des Aortendurchmessers bei Storungen der Blutdruckregulation Wien klin Wchnschr, 45 1248, 1932
Zdansky, E Zur Kritik der Kreuzfuchsschen Aortenmessung Fortschr a d Geb d Röntgenstrahlen, 45 40, 1932
Zdansky, E Rontgenuntersuchung des Herzens Wien klin Wchnschr, 46 432, 1933
Zdansky, E Ueber des Rontgenbild des Lungenodems, gleichzeitig ein Beitrag zur Frage der Pathogenese des Lungenodems Rontgenpraxis, 5 248, 1933
Zdansky, E Was leistet der Rontgenuntersuchung fur die Diagnose der schwieligen Perikarditis? Wien klin Wchnschr, 46 993, 1933
Zdansky, E Ueber die Veranderungen der Herzgrosse und form nach einmaliger Arbeitsleistung Ztschr f klin Med, 131 112, 1936
Zdansky, E Rontgenbefunde des Herzens bei Hochdruckheiten Radiol Rundschau, 5 281, 1937
Zdansky, E Rontgenologisch diagnostizierte verkalkte Milzzyste Rontgenpraxis, 13 66, 1941
Zdansky, E Das Syndrom der Herzvergrosserung beim Myxodem des Erwachsenen Wien med Wchnschr, 56 46, 1943
Zdansky, E Rontgenologisches zum Sportherzproblem Wien klin Wchnschr, 59 393, 1947
Zdansky, E Die Beurteilung der Herzkammern im Rontgenbild Klin Med, 3 132, 1948
Zdansky, E Rontgenologische Einblicke in die Funktion des Emphysemherzens Radiologia Austriaca, 3, 1950
Zdansky, E Rontgenologie des Lungenkreislaufs Verh d deutsch Ges f Kreislaufforschg, 1951
Zdansky, E Die Rontgendiagnostik der Insuffizienz des Cor pulmonale und Cor hypertensicum Nauheimer Fortbildg, 1951, 16
Zdansky, E Zur Rontgenologie der Dynamik des Herzens Fortschr a d Geb d Rontgenstrahlen, 75 179, 1951.
Zdansky, E Cor triloculare biatriatum mit Pulmonalstenose im Rontgenbild Wien klin Wchnschr, 63 144, 1951
Zdansky, E Die Funktion des Herzens im Rontgenbild Fortschr a d Geb d Rontgenstrahlen, 76 275, 1952
Zdansky, E, and Ellinger, E Zur Frage der Haufigkeit der Hiatushernien und ihrer Beziehung zur Angina pectoris Med Klin, 29 47, 1933.
Zdansky, E, and Ellinger, E Rontgenkymographische Untersuchungen am Herzen I Fortschr a d Geb d Rontgenstrahlen, 47 648, 1933
Zdansky, E, and Ellinger, F Röntgenkymographische Untersuchungen am Herzen Wien med Wchnschr, 49 240, 1934
Zehbe, M Beobachtungen am Herzen und der Aorta Dtsch med Wchnschr, 42 315, 1916
Zehbe, M Beitrage zur Rontgenuntersuchung des Herzens Fortschr a d Geb d Rontgenstrahlen, 26 424, 1917
Zehbe, M Ein Fall von Panzerherz Fortschr a d Geb d Rontgenstrahlen, 30 32, 1922–3
Zeidler, H Drei Falle von congenitalem Defekt der Vorhofscheidewand Arch f klin Med, 131 85, 1920
Zeisler and Bloomfield, E Trikuspidalstenose Uebersicht die Literatur mit Bericht uber einen Fall mit intravitaler Diagnose Am Heart J, 8 138, 1935
Zezschwitz, P Zur Bewegung der Basis der hinteren Herzkammerwand und des linken Vorhofes Munchen med Wchnschr., 71 614, 1924
Zimmermann Zur Klinik und Pathologie der Pulmonalsklerose. Wien Arch. inn Med, 24 301, 1934

Zins, B Zwei Falle von Schwangerschaft bei Ductus Botalli persistens Wien klin Wchnschr, *40* 1227, 1927

Zins, B, and Rosler, H Kasuistischer Beitrag zur Beeinflussung des Myxodemherzens durch Thyreoidin Wien klin Wchnschr, *39* 1360, 1926

Zondek, H Eine Methode zur Messung der Herzgrosse im Rontgenbild. Med Klin, *12* 147, 1918

Zondek, H Das Myxodemherz Munchen med Wchnschr, *55* 1180, 1918, *56* 681, 1919

Zondek, H Herzbefunde bei Leuchtgasvergifteten, Dtsch. med. Wchnschr., *45* 678, 1919, *46* 235, 1920

Zweifel, C Der Zwerchfellhochstand beim Lungeninfarkt Fortschr. a d Geb d. Rontgenstrahlen, *52* 222, 1935

van Zwaluwenburg, J G A plea for the use of fluoroscopy in the examination of the heart and great vessels. Am J Roentgenol, 7 1,1920

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Zins, B Zwei Falle von Schwangerschaft bei Ductus Botalli persistens Wien klin. Wchnschr, *40* 1227, 1927

Zins, B, and Rosler, H Kasuistischer Beitrag zur Beeinflussung des Myxodemherzens durch Thyreoidin Wien klin Wchnschr, *39* 1360, 1926.

Zondek, H Eine Methode zur Messung der Herzgrosse im Rontgenbild. Med Klin, *12* 147, 1918

Zondek, H Das Myxodemherz Munchen med Wchnschr, *55* 1180, 1918, *56* 681, 1919

Zondek, H Herzbefunde bei Leuchtgasvergifteten, Dtsch med Wchnschr, *45* 678, 1919, *46* 235, 1920

Zweifel, C Der Zwerchfellhochstand beim Lungeninfarkt Fortschr a d Geb d Rontgenstrahlen, *52* 222, 1935

van Zwaluwenburg, J G A plea for the use of fluoroscopy in the examination of the heart and great vessels Am J Roentgenol, 7 1,1920

van Zwaluwenburg, J G, and Warren, L F The diagnostic value of the orthodiagram in heart disease Arch Int Med., 7 137, 1911

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